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The Bancroft Memorial Lecture.[1]

THE NERVOUS FACTORS IN DISORDERS OF THE HEART.

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I WISH in the first place to say how highly I appreciate the compliment you have paid me in inviting me to deliver the Bancroft Oration this year.

When your predecessors decided upon this very graceful method of keeping green the memory of your renowned physician, I feel sure that they had in mind that some account of the man whose gifts to medicine it commemorates should be an integral part of the oration.

Joseph Bancroft enrolled as a student at the Manchester Royal Infirmary at a time when the universities offered so little encouragement to those interested in natural science that it had become customary for such men to embark upon a medical career largely with the idea of thus providing the means for indulging their scientific tastes.

Graduation found them seeking opportunities for practising in an environment where they could follow their particular bent, and it is not surprising that so many of them found their way to the less known parts of the Empire where there were wide fields of natural history still unexplored.

We can have little doubt that this was the lure that attracted young Dr. Bancroft to Brisbane in 1864. Dr. Sandford Jackson[(1)] in his historical notes from the records of the Brisbane Hospital has given us a graphic account of how Joseph Bancroft indulged his flair for botanical research at his experimental farm at Deception Bay. Here he turned his scientific knowledge to great practical service for his adopted country, breeding rust-proof wheats, hybridizing fruits, and exhibiting such a fund of botanical lore that it is perhaps surprising that he did not, like Darwin and that other Joseph so well known in Australian history, Sir Joseph Banks, forsake medical practice altogether.

Fortunately his interest in medicine was a very real one, and he was soon immersed in a large general

[1] Delivered at Brisbane, June 1, 1934.

practice in which he must have often deplored the lack of opportunity to carry out investigations on his patients comparable to those he was conducting on his farm. We can well imagine how his scientific interest would be aroused by learning that microfilariæ had been discovered in one of the cases of lymphatic obstruction that he was finding so puzzling in his practice. Here right to his hand was the kind of problem he was specially suited to solve, and he immediately plunged enthusiastically into a study of the disease and, in April, 1877, was able to announce that he had discovered the parent filarial worm, and thus supplied the missing link in the life history of this widespread tropical helminth.

While in paying our homage to Joseph Bancroft tonight our thoughts will naturally dwell upon the discovery that placed the name of this Queensland general practitioner so high on the list of famous British investigators of tropical diseases, but we must not forget the numerous less spectacular researches in which his life was spent.

In these days when research is so apt to be regarded as the special prerogative of the whole-time laboratory worker, those on whom the lot has fallen to see disease in its natural environment may well draw inspiration from the thought that the research through which Joseph Bancroft chiefly achieved his fame, was carried out by him in his own home amid the litter and distractions of a large general practice.

I deeply regret the great loss this Branch has sustained since the Oration was delivered last year in the death of Thomas Lane Bancroft. Ably treading in the footsteps of his distinguished father, Thomas Bancroft not only carried out further important investigations into the life history of *Filaria bancrofti*, but also conducted many biological and botanical researches of great economic value to this State. I should like to take this opportunity of conveying, through you, my very sincere sympathy to Mrs. Bancroft and her family.

I have chosen "The Nervous Factors in Disorders of the Heart" as the somewhat clumsy title for my address to you tonight because I have found that, while there is a very general recognition that cardiac disorders may be of purely nervous origin, there is by no means the same appreciation that the symptoms for which patients having organic disease of the heart consult us are often of precisely the same character.

It is my purpose to review briefly our present knowledge of the nervous relationships of the heart and to endeavour to correlate this with clinical experience in regard to those cardiac disorders in which the nervous factors appear to play a predominant part.

The selection of the heart as the seat of the emotions in ancient times was probably the earliest recognition of its ready response to nervous influences, and since the dawn of scientific medicine physicians have been confronted with the difficulty of differentiating between its organic and nervous disorders.

The extensive researches carried out in recent years in regard to all phases of the circulation have greatly advanced our knowledge and compelled us largely to recast our ideas about many of its aspects. The invention of the ink polygraph was the first real step towards a proper understanding of the arrhythmias, while the electrocardiograph, which so quickly followed in its wake, not only demonstrates any arrhythmia present, but often enables us to detect structural changes in the musculature of the heart at a stage when these can be at best suspected as the result of other means of clinical observation.

Experiments carried out on the isolated heart have greatly clarified our ideas as regards the functions or the cardiac muscle and shown how modifications in the rhythm can be brought about by external stimuli.

Research into the nervous connexions of the heart and experimental stimulation in various directions have demonstrated how intimate is the relationship between nerve and muscle and how the conductivity can be enhanced, retarded and distorted through nervous influences, and, further, how the whole nutrition of the heart may be modified as the result of the abundant nerve supply to the coronary vessels.

More precise knowledge of the afferent pressor and depressor nerves passing from the aorta and carotid sinus to the vasomotor centre in the medulla has brought home to us the need to envisage the circulatory mechanism as a whole since the heart's activity can be so profoundly modified through the changes in vascular tonus initiated by these nerves.

In several recorded cases the carotid sinus has been found so irritable that digital pressure induces profound syncope and even convulsions, while Soma Weiss and James P. Baker,[2] in an electrocardiographic study of such cases, found that under varying degrees of pressure such variations in rhythm as extrasystoles, auricular fibrillation and complete heart block could be produced.

Of extreme interest are the results of biochemical researches which have revealed that the control over muscular contraction by involuntary nerves is exercised by virtue of the liberation of a chemical body which is actually the effective agent in producing the characteristic response. Thus stimulation of the vagus liberates a chemical body, probably identical with acetyl choline, which has an inhibiting action on the cardiac mechanism varying in degree from a slight slowing to abolishing conduction altogether so that heart block or even arrest of the ventricle may result. Stimulation of the sympathetic nerves, on the other hand, has been shown to generate, at least at the vascular neuro-muscular junctions, an adrenal-like substance which has been named sympathine.

Reference may here be made to a report by Dr. W. G. Hume[3] on the effect of the intravenous injections of two and a half minims of one in 1,000 solution of adrenaline chloride into three healthy persons whose electrocardiograms were normal. In one case the normal rhythm was entirely replaced by ventricular complexes, in another complete auricular ventricular dissociation resulted, while the third exhibited auricular fibrillation.

Though the reactions have no necessary relationship, the close similarity between these adrenaline effects and those resulting from pressure upon an

irritable carotid sinus are particularly interesting in view of the well established fact that afferent nerves from the aorta and carotid sinus to the vasomotor centres instantly affect the output of adrenaline.

While these recent accretions to our knowledge have greatly enhanced our understanding of the autonomic working of the circulation, we are still much in the dark in regard to its points of contact with the central nervous system, which must primarily provide for modification in various directions under condition of emergency and for conscious appreciation of serious cardiac inefficiency. Thus we find that under conditions of emotional stress the circulation, though detached from voluntary control, can be profoundly affected by stimuli projected in some way from the higher centres. Owing, however, to the dual character of the cardio-vascular innervations, appreciable change can obviously be produced only by disturbing the balance between the vagus and sympathetic through augmentation or inhibition of one or other.

Regarding emotions as effective aspects of instinctive reactions, we find that, while the instinctive reactions of the circulation can be accurately forecasted in various types of animals, in man this is far from being the case, an anomaly best explained on a hypothesis of a congenital or acquired disbalance between vagus and sympathetic, and an attempt has been made to classify man according to behaviour as either vagotonic or sympatheticotonic.

On this basis we may perhaps explain why in moments of ecstacy one poet finds his heart stand still, while another records his racing pulses. The danger instinct again finds one individual under sympathetic and adrenaline stimulation equipped for flight or aggression with tightened vessels and bounding heart, and another with vagus in command collapsed in what Rivers[4] regards as a complex distortion of the immobility reaction so well known in insects and animals.

While the exact course of these emotional stimuli which play so prominent a part in nervous affections of the heart has not been defined, there is good reason to suspect that they primarily act through the vasomotor centre in the medulla.

Of the various theories offered to explain conscious appreciation of disorders of viscera devoid of sensory nerves the hypothetical viscero-sensory reflex of James Mackenzie[5] is the most satisfactory. It is perhaps unnecessary to remind you that this theory is based on the assumption that under normal conditions the constant stream of afferents passing to the spinal cord from a viscus merely makes contact with the efferents controlling its own blood vessels, muscles, secreting glands *et cetera*, but that under morbid conditions the intensity of the afferents is so enhanced that their effects overflow into neighbouring centres and stimulate sensory nerves conveyed by corresponding posterior nerve roots and sometimes, in addition, motor cells, when contraction of corresponding skeletal muscles results. Mackenzie[6] further suggests that as a result of continuous stimulation such centres tend to become modified in some unknown way into what he calls an irritabie focus, so that the intensity of impulses reaching it become enhanced, leading to hyperalgesia of the corresponding segment of the body, with increased tonicity of the muscles. A further complexity arises from the tendency for stimuli reaching the focus from various directions to provoke the response normally only aroused by the diseased viscus.

Quite apart from the viscero-sensory reflex there is an intangible consciousness of the heart's action which plays an important part in the nervous disorders of the heart owing to the powerful suggestion of morbidity that the unusual sensations engender. Most commonly the awareness of the heart's action is described as palpitation, a vague term in popular use which by no means necessarily implies any alteration in rate or regularity. Indeed, quite often it can be described only as consciousness of the beating of a perfectly normal heart, an experience often associated with mild gastric disturbances. In other cases, however, the term may cover appreciation of any variation of rate of rhythm, including tachycardia, extra systoles, and even auricular fibrillation. Quite often, however, when definite irregularities are present their nature is fairly accurately appreciated, ectopic beats being interpreted as the heart turning over and fibrillation as a state in which the heart alternately turns over and races.

One patient, quite unaware that he had had a coronary occlusion, described how on several occasions while lying in bed he felt that the blood was trying to get into his heart through too narrow an opening till one night it seemed to get blocked altogether and, though he had no pain, he became cold and clammy and felt he was going to die.

Mackenzie states that people with sinus arrhythmia are never conscious of it, but in my experience adults exhibiting this type of rhythm are frequently heart conscious and complain of palpitation.

The nervous relationships involved in these reactions are at present unknown. In the simple palpitation brought on by such causes as mental worry, late suppers, and tobacco, the throbbing complained of is often associated with marked relaxation of the arteries, and this probably explains to some extent the thumping sound experienced when the ear rests on the pillow. This hyperacusis may be the most distressing feature of the attacks, and I have more than once been consulted by patients complaining of peculiar auditory sensations described as something between an intermittent whisper and a "swishing" sound which has proved to be due to unrecognized extrasystoles.

In cases of irregularity, concentration of attention on the heart often plays an important part, and many patients with ectopic beats and auricular fibrillation first become conscious of them after their discovery at a medical examination.

The influence of suggestion was well illustrated in an ex-soldier who complained of palpitation and a sensation of his heart scraping against something with every beat, but who improved rapidly after X rays had shown that a piece of shrapnel previously reported to be lying against his heart was really deeply embedded in the liver.

Despite extensive clinical and pathological research, no clear-cut explanation is yet forthcoming that will account for the great variations in threshold level of stimulation met with in different individuals.

While in some cases abnormal irritability would appear to be a congenital defect, and in others it

seems to result from certain forms of nervous stress, there is much clinical evidence that the prolonged action of various toxic agencies is the chief underlying cause, and the term psychotoxic heart has recently come into use.

The fact that from time to time patients, who have for years appeared to suffer from this type of disorder, develop frank evidence of organic changes, particularly coronary occlusion, suggests that some, at least, of these toxins may in time produce structural alterations.

On the other hand, owing to the prolonged nature of the illness it is difficult to exclude pure coincidence as the explanation of such exceptional cases.

It may, however, be of interest to mention that among the cases of coronary occlusion seen by me in comparatively young people presenting very little evidence of general cardiovascular disease there have been two ex-soldiers under treatment since the Great War for "D.A.H." (disordered action of the heart) or the effort syndrome, while four others were heavy smokers who gave a definite history of having at various times had to abandon temporarily the use of tobacco on account of paroxysms of tachycardia and precordial discomfort.

The ordinary fainting attack furnishes an excellent objective illustration of the extraordinary maze of pathways traversed by stimuli competent to bring about disorder of the heart. Actually, as Sir Thomas Lewis[7] has so clearly demonstrated, the most common form of syncope or fainting is a vasovagal phenomenon in which vasomotor changes and vagus slowing of the heart are combined in varying degrees.

It is important to remember that syncope is just as much a nervous phenomenon, when it occurs in people with functional or organic murmurs, as it is in those whose hearts present no abnormality. Unfortunately, those most liable to such attacks—people of a nervous habit, those in poor general health, and rapidly growing girls in their late 'teens—belong to groups in which such murmurs are especially likely to be found, and it is by no means unusual to see patients, especially young women with mild degrees of mitral stenosis, who have been sentenced to lifelong invalidism as a punishment for fainting.

Probably the most frequent immediate precursor of syncope is standing, and it appears that in such cases relaxation of the splanchnic veins associated with a stream of afferents from the alimentary tract plays the chief part. Fainting after an enema or profuse evacuations is, no doubt, of the same character, and I was once asked to see a patient with nervous colitis who, though lying in bed, had a really alarming syncopal attack each time he used the bed pan. It may be of interest to add that the condition was relieved by the injection of adrenaline just before each evacuation. Having in mind the aortic vago-depressor nerves, it is perhaps not surprising that an aortic lesion is the only type of structural disorder of the heart in which syncope is regarded as having a sequential relationship.

Sir Thomas Lewis drew attention to the many points of resemblance between the common types of syncope and that occurring on pressure over a hypersensitive carotid sinus, and it is possible that sinus pressure may explain such unexpected syncopal attacks during anæsthesia as in an alarming personal experience in which at the very inception of ether anæsthesia, so complete and prolonged asystole took place that the surgeon present made a confident pronouncement of death.

Simple tachycardia is so familiar a phenomenon that we are prone to forget how little we actually know of its nervous relationships.

In the case of the rapid pulse that results from heart failure, some at least of the underlying factors are known, for it has been established that the anoxæmia that results has an accelerating action, and also that nerves connecting the right auricle and great veins with the medulla, when stimulated by a rising venous pressure, bring about vagal depression and sympathetic stimulation.

On the other hand, we know practically nothing about the nervous relationships that are made use of when what we may speak of as extracardiac nervous stimuli, aroused by the emotions or activated in some way by toxic agencies, increase the heart rate.

In actual clinical practice careful discrimination combined with common sense is often called for in determining the real cause of a persistently rapid pulse, and the consumption of digitalis would fall enormously if it was more generally realized that an increase in the rate is never the only sign of heart failure.

Those suffering from valvular lesions of rheumatic origin are particularly subject to this nervous type of tachycardia, and the initiation of treatment for a failing heart instead of for the nervous disturbance tends to exaggerate rather than improve the condition.

As illustrative of this point I may refer to the case of a nervous typist with mitral stenosis whose tachycardia had persisted after spending a month in bed on digitalis, but promptly settled down when she went away for a holiday armed with a bromide mixture.

This extrinsic type of acceleration is often, however, a serious embarrassment in the graver forms of heart disease where owing to myocardial damage the reserve is seriously curtailed, and the prospects of postponing disaster will often depend upon the possibility of its control.

Quite commonly timely recognition and elimination of an unsatisfactory emotional environment will do more than all our other measures to restore some measure of equilibrium to patients in a state of congestive heart failure. I would remind you also of how much the prognosis in thyreotoxicosis is influenced by the state of the myocardium at the time it develops, and also of the disappointment that so often dogs our efforts to treat the vicious circle that forms when a nervous tachycardia harasses a heart in the vascular degenerative stage of hypertensive disease.

The fact that every known type of cardiac arrhythmia can be produced in normal hearts by such means as electrical stimulation, pressure on an irritable carotid sinus, or an injection of adrenaline, should serve to dissipate the widely held suspicion that these irregularities are necessarily indicative of myocardial disease.

While it is now very generally recognized that sinus arrhythmia merely records variations in vagal tone

and has no pathological significance, I find that I have recorded its presence so often in cases filed as "nervous affections of the heart" that I feel that such vagus excitability in adults may be taken as suggestive of nervous instability. The dictum of Sir James Mackenzie[8] that the presence of this irregularity generally speaking points to an intact myocardium, while a useful guide, must not be too blindly accepted, for I have seen recently an electrocardiogram in which both this arrhythmia and the changes characteristic of a recent coronary occlusion were clearly demonstrated.

The presence of extrasystoles or ectopic beats can be interpreted only as evidence that another part of the heart is for the time being more excitable than the normal starting point of the cycle. How such exciting factors as an attack of indigestion or an extra cigar act is not clear, but there is abundant evidence that it is not by producing any recognizable pathological lesion.

The readiness with which extra systoles appear in the latter period of life suggests that minor deficiencies of blood supply may be a factor in these cases. Like Mackenzie,[8] I have watched so many people pass from middle to old age while harbouring this irregularity that I fully endorse his dictum that "when the extrasystole is the only abnormal sign, the prognosis is a favourable one, and when it is associated with other signs, the prognosis is to be based upon the other signs". It should, perhaps, be noted that extrasystoles often coexist with auricular fibrillation, and that when the rate is a slow one, it may be almost impossible to determine the nature of the arrhythmia without the help of an electrocardiogram.

The close relationship between the special disturbances of the rhythm of the auricles, paroxysmal tachycardia, auricular flutter and auricular fibrillation is shown by the readiness with which each can be produced in sound hearts by electrical stimulation, by the fact that each may occur in paroxysms, and, further, by the fact that, especially in the paroxysmal form, each can usually be controlled by quinidine.

While it is generally realized that paroxysmal auricular tachycardia is a nervous disturbance, the essentially nervous character of auricular flutter and of auricular fibrillation is apt to be overlooked, and these conditions are rather regarded as an integral part of the structural disease with which they are so often associated.

Paroxysmal auricular flutter, owing to the partial heart block which accompanies it, probably often passes unrecognized, for it is often of quite a transitory character.

In a case recently seen flutter accompanying thyreotoxicosis promptly disappeared after subtotal thyreoidectomy, but soon recurred with hypertrophy of the remaining gland, disappeared again after a second operation, only to reappear later in association with a high metabolic rate, but finally disappeared after a third operation.

Paroxysmal auricular fibrillation in many ways suggests paroxysmal tachycardia gone wrong. Seemingly what goes wrong is the intrinsic conducting mechanism of the auricle, but it seems probable that this may at times be a purely functional disorder for, though paroxysmal fibrillation is more likely to affect a damaged heart, it quite often appears in an apparently healthy one. On the other hand, owing to the disorderly character of the rhythm, heart failure is liable to set in much earlier than in paroxysmal tachycardia. John Cowan,[10] in discussing auricular fibrillation, summarizes the position by stating that "paroxysmal fibrillation may be due to the action of various intoxications which, while more likely to affect an already damaged heart, may apparently also affect a healthy organ".

In my personal experience examples of the arrhythmia have been seen under such varying conditions as at the onset of pneumonia, lasting less than twenty-four hours, in an apparently healthy man who made a good recovery.

In a plump robust woman the arrhythmia, lasting a few hours, followed an attack of flatulent indigestion, and she stated she had had several similar attacks.

In a man, who subsequently developed the persistent type, the paroxysms were apparently related to tobacco, for he remained free for six months when not smoking, but then had an attack after smoking six cigarettes.

In a chronic asthmatic a first attack came on with a paroxysm of asthma and had persisted for five days in spite of free exhibition of digitalis, when quinidine was given and brought about rapid restoration of normal rhythm and disappearance of the œdema and signs of heart failure which had begun to appear. The rate was maintained at about 80, and the patient remained perfectly well during further observation. The electrocardiogram taken when he was convalescent revealed no abnormality.

While in the more chronic type of auricular fibrillation the prognosis depends mainly upon the structural disease usually associated with it, due consideration must be given to the nervous temperament of the individual, for at times no maintenance dose of digitalis will prevent paroxysms of disabling tachycardia being brought on by emotional stimuli.

Though experimental studies have clearly demonstrated the nerve relations of heart block, the chronic type usually met with clinically is almost always associated with structural changes.

That it is not a disease entity but a disturbance of function we have, however, clinical evidence in the many recorded cases in which transitory types of block, most often incomplete, occur. These cases are seen in such conditions as typhoid fever, pneumonia, influenza and even obstructive jaundice, when recovery of the disease is usually accompanied by restoration of the normal rhythm.

Even in the presence of gross structural disease transitory block may occur, and, if immediate disaster can be avoided, may completely pass off. It is well known that digitalis and quinidine may produce complete block in such hearts, but purely nervous stimuli, especially those arising from the gastro-intestinal tract, may do the same, and I have seen both partial heart block and left branch bundle block converted quite temporarily into complete block by gastric disturbances following indiscretion in diet.

The nervous factors concerned in the affective side of cardiac disorders are so numerous and complex that it will be convenient to limit consideration of subjective symptoms to a study of actual pain on the left side of the body in the area of distribution of the first four dorsal nerves.

Of the many pains experienced in this region the anginal syndrome with its motor and sensory components is the only one of clearly cardiac origin that can, *per se*, be regarded as of prognostic significance, and any pain which does not present these dual characteristics should be very carefully scrutinized before being accepted as evidence of cardiac embarrassment.

The vague pain and tenderness in the precordial region so often met with in nervous or dyspeptic young people with valvular defects, are explained most satisfactorily on the basis of extrinsic stimulation of the irritable focus postulated by Mackenzie,[6] and much unhappiness and unnecessary invalidism would be avoided if this were better recognized, and if treatment in so far as the heart is concerned were guided solely by the measure of its efficiency.

The explanation of similar pains that, though less common, are at times complained of by nervous people with quite normal hearts, is more difficult; but there seems to be some overlapping of the areas of pain referred from the heart and upper abdominal viscera, despite the different levels at which their afferents enter the spinal cord.

Careful investigation should trace to their source pains arising from direct stimulation of the sensory nerve endings in the thoracic walls, as in such conditions as eroding aneurysms or mediastinal growths, traumatic arthritis of a costo-chondral junction and diaphragmatic pleurisy; I have, however, seen all of them diagnosed as *angina pectoris*. Much more difficulty may be experienced when pain results from direct irritation of the nerve roots, for in such cases muscular spasm is commonly present, as in the following case.

A man aged thirty-seven years was subject for fourteen months to recurring attacks of gripping pain in the left side of the chest extending into the left arm, particularly when sweeping his yard or turning in bed. An X ray examination revealed marked spondylitis of the upper dorsal and cervical region, but there was no clinical, radiological or electrocardiographic abnormality in the heart.

Of similar origin are, no doubt, many of the attacks, described in Compensation Courts as heart strain, in which violent thoracic pain accompanied by tachycardia and heart consciousness results from severe muscular effort in individuals with apparently normal hearts.

In one such case in which my evidence, while it failed to convince the judge, had the amusing sequel of bringing the successful plaintiff to me for treatment, there was not only well marked spondylitis, but also gross oral sepsis.

The occasional appearance of more or less typical attacks of *angina pectoris* in individuals with apparently normal hearts is more difficult to explain, but the number of such cases has greatly diminished since the introduction of the electrocardiograph.

On the rare occasions when the classical syndrome is met with in quite young people it is possible that an associated psychic instability and functional dyspepsia may in some way set up a reflex spasm of normal coronary vessels. In the case of older people, however, even a normal electrocardiogram will not exclude the possibility of some coronary narrowing that has not yet caused any definite myocardial damage, and in two cases of my own a coronary occlusion took place soon after a normal electrocardiogram had been taken.

The special interest that the anginal syndrome has so long aroused on account of its dramatic character and sinister implications has been greatly enhanced since the electrocardiograph has established its frequent relationship with coronary occlusion. If further excuse for discussing its nervous relationship were needed, it would surely be found in the alarming increase in the number of deaths recorded in recent years under the heading of *angina pectoris* and coronary occlusion.

Through the courtesy of the Government Statistician of New South Wales I have with me the figures dealing with the deaths that have taken place from these causes in each year since 1924. They are set out in the accompanying table.

These statistics show that in New South Wales the deaths under the heading "*Angina Pectoris*" have risen from a total of 106, or 47 per million of population, in 1924 to a total of 262, or 100 per million of population, in 1933, while at the same time deaths recorded as from diseases of the coronary arteries have risen from a total of 34, or 15 per million of population, in 1924 to 432, or 166 per million of population, in 1933. While admitting that allowance must always be made for improved diagnosis and a change of fashion in terminology, it is hard to believe that this alone could explain, in relation to so striking a syndrome, an increase in

TABLE I.

Year.	Mean Population.	(a) *Angina Pectoris.*				(b) Diseases of Coronary Arteries.				Sudden Death (including Syncope and Sudden Heart Failure.)		
		Males.	Females.	Persons.	Rate per Million of Population.	Males.	Females.	Persons.	Rate per Million of Population.	Males.	Females.	Persons.
1924	2,244,471	78	28	106	47	16	18	34	15	9	4	13
1925	2,295,605	73	27	100	44	19	18	37	16	5	4	9
1926	2,347,014	101	53	154	66	29	30	59	25	11	3	14
1927	2,404,016	145	54	199	83	22	28	50	21	9	3	12
1928	2,460,568	171	58	229	93	27	25	52	21	6	4	10
1929	2,508,208	168	71	239	95	46	12	58	23	3	–	3
1930	2,532,497	183	72	255	101	62	38	100	39	1	–	1
1931	2,556,097	156	61	217	85	175	70	245	96	4	1	5
1932	2,579,983	186	68	254	98	189	91	280	109	5	2	7
1933	2,602,037	180	82	262	101	298	134	432	166	10	–	10

the combined figures of *angina pectoris* and coronary occlusion during a period of ten years of from 140 to 698.

Of considerable interest from the economic point of view is the comparatively early age at which coronary occlusion occurs. No general statistics on this point are available, but my own records show that of forty-seven patients who attended at my consulting room whose electrocardiograms were typical of coronary occlusion over 70% were under sixty years of age, and that of the total almost 50% were between the ages of fifty to fifty-nine. It is interesting to note, as showing that our former standards of differentiation between *angina pectoris* and coronary occlusion can no longer be maintained, that in a considerable number of these ambulatory cases of occlusion there was no history of severe or sustained pain, and without the aid of the electrocardiograph the patients could only have been diagnosed as suffering from *angina pectoris*.

In considering the nervous relationship of the angina syndrome, it is as well to keep in mind that these may be somewhat different when frank aortic disease exists since the relative parts played here between structural restriction of the flow into the coronary vessels and a possible alteration of the functions of the aortic depressor nerves leading to constriction of the coronary vessels instead of a more normal dilatation is still the subject of controversy.

The long recognized extracardiac relationships of *angina pectoris* have attracted still greater attention since coronary thrombosis has come to be regarded as its probable sequel. The more closely this aspect is studied, the more evident it seems that the anginal syndrome results from vascular spasm imposed upon vessels progressing through various stages of degeneration towards ultimate closure.

In the absence of any direct means of investigating the coronary circulation, it is obviously hazardous to attempt to forecast the imminence of an occlusion, but some guidance may be obtained by a careful review of the nervous surroundings backed by electrocardiographic findings. Thus it is so well recognized that those of a highly emotional temperament may experience anginal seizures over many years and that a satisfactory electrocardiogram in such cases may well justify some measure of optimism.

In my personal experience afferents arising from gastric and duodenal ulcers and also from the gall-bladder have seemed to be the chief disturbing factor in some prolonged cases, and treatment of the lesion has resulted in marked improvement.

Thus in two cases, despite myocardial defects and hypertension, the removal of gall-stones was followed by remarkable freedom from almost intolerable angina till occlusion took place in one a year, and in the other over two years later.

Again a man whose angina, chiefly on effort, woke him very constantly about two in the morning, though he has quite definite myocardial changes, has had little trouble from his attacks for nearly five years since receiving treatment for a duodenal ulcer.

The part played by irritation of the posterior roots in relation to spondylitis is apt to be overlooked but is, I think, an important factor in the early initiation of vascular spasm. In several cases with a long history of angina on exertion, attacks brought on by actions involving movement or fixation of this part of the spine, such as turning the head, looking upwards, sneezing and driving a motor car, have suggested that the well established spondylitis demonstrated by the X rays is a contributing factor.

In phlegmatic and unemotional individuals, on the other hand, angina and occlusion come in such swift succession that after even an apparently mild attack an electrocardiogram rarely fails to show that thrombosis has already taken place, while it is especially in such people that sudden death or desperate jeopardy comes without the slightest warning.

When we attempt to interpret the advanced coronary disease so often found *post mortem* in such cases in terms of Mackenzie's viscero-sensory reflex we are forced to the conclusion that something more than a stream of afferent impulses from a morbid viscus is needed to activate his suggested irritable focus, and that it can function only when the general threshold level of stimulation is low.

This conclusion is supported by experience after occlusion occurs, for these placid patients are little troubled by pains after the first few stormy days, and but rarely complain of it after recovery.

In nervous and irritable individuals, on the other hand, recurring pains and vasomotor phenomena brought on by excitement, by transitory flatulence, by constipation, by even the late arrival of the doctor, may for weeks create an atmosphere of anxiety and fear that the thrombosis is extending. In these, too, for long after a tardy convalescence, spasms of retrosternal pains may be readily induced.

When, however, as sometimes happens in these nervous patients, complete disappearance of long standing angina follows recovery from an occlusion, a strictly limited area of disease is suggested.

Such must surely have been the case in a man of sixty-six, now in excellent health, whose angina of some years duration culminated in a typical coronary occlusion in 1924, and who has remained so completely free from symptoms since that he was able to sustain fractures of a leg and an arm in a motor accident last year without recurrence.

Possibly the less ostentatious disappearance of angina occasionally observed in younger people may have the same explanation.

That quite young people do suffer from coronary occlusions was well demonstrated to me recently when a nursing probationer of nineteen, now recovered, sustained a classical attack within a week or two of resuming duty after a mild attack of scarlet fever.

In now concluding may I express the hope that even though you may feel that I have told you nothing that is new, you will at least agree that the interpretation of the nervous relationship of disorders of the heart is one of the most difficult and at the same time most interesting of the problems with which a physician is confronted, and further, that it is only by carefully marshalling all the available data and bringing to bear on them a judicial mind backed by experience and common sense that mistakes can be reduced to a minimum.

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SOME REMARKS ON THE DIAGNOSIS OF ACUTE CONDITIONS OF THE ABDOMEN.[1]

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THE purpose of this short paper is merely to open the subject that has been chosen for this evening's discussion; and therefore it is obvious that one can touch only the fringe of such a comprehensive subject as the diagnosis of acute conditions of the abdomen. Few subjects in abdominal surgery have received so much attention in text books and journals; and it would be presumption to endeavour to add to what has already been written on the differential diagnosis of these conditions. I am assuming that the term acute conditions of the abdomen designates conditions which are to be regarded as requiring urgent surgical interference. Regarded in that light, our first object is to be able to determine the necessity for operation. The final differential diagnosis of the cause, though of the utmost importance, must take second place if we are to diminish the mortality of some of the conditions to be described later. Therefore, it is my purpose to mention one or two points which may help in determining whether an abdominal lesion demands immediate exploration. To perform an urgent laparotomy in a case in which subsequent examination shows that it was unnecessary is admittedly bad surgery, and is an admission of diagnostic failure; but I believe that the mortality rate of certain conditions described later will not be reduced unless we are prepared to perform a laparotomy sometimes without waiting to make a detailed diagnosis and are even prepared to risk an unnecessary laparotomy rather than disregard the suspicious signs that may indicate the early stages of a condition convertible by delay into a disaster. I shall refer to these suspicious signs later.

Acute appendicitis, as an acute condition of the abdomen, we have always with us; but I do not propose to delay on that subject. Dr. Pomroy has recently given you an admirable paper on certain aspects of this condition, and, within certain limits, I think all of us agreed with his opinions. He has published some very striking figures, and I think it is not inopportune to draw your attention to some other figures. Table I gives the statistics for the admissions to the Adelaide Hospital for the last three years of patients suffering from acute appendicitis.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on February 22, 1934.

TABLE I.
Acute Appendicitis.

Year.	Total Cases.	Deaths.
1931	279	8
1932	293	3
1933	329	2
Total	901	13

Mortality: 1·3%.

Admittedly there are many fallacies in any series of statistics, and at the Adelaide Hospital no allowance is made for the subacute case. But, none the less, considering that the care of these patients has been shared between at least ten surgeons, the figures will compare favourably with any others; and I believe that the low mortality rate disclosed by them is due to three factors: (i) The general public is awake to the possibilities of acute abdominal pain and seeks advice early. (ii) The general practitioner makes an early diagnosis and sends the patient into the hospital early. (iii) The majority of these patients are therefore seen early by the honorary staff, often within the first twenty-four hours, and are submitted to early operation.

We may perhaps feel that the mortality, 0·6%, for the last year is satisfactory. Unfortunately there are several other conditions which afford us no such complacency. In the statistics of the Adelaide Hospital for the last three years two groups of cases show the need for much earlier diagnosis and even for earlier laparotomy in the absence of a complete diagnosis. The two groups are: (i) perforations of viscera, (ii) intestinal obstruction associated with internal strangulation. And of these two I am most concerned with the second.

From the first group I have excluded the traumatic perforations, and from the second group the cases of strangulation in an external hernial orifice. It is notorious that the diagnosis of traumatic perforations may be very obvious, or very obscure, and they are often so complicated by injuries to other systems. But in them and in external strangulations the history is so compelling that the condition in question is immediately suspected, though in traumatic cases it may be some time before one has the courage of one's convictions. It is easy to suspect, but if you suspect you should be prepared to go the whole way without waiting. It will mean making some mistakes, but it will save lives otherwise jeopardized by delay.

TABLE II.

Ruptured Duodenal Ulcer.

Year.	Total Cases.	Deaths.
1931	26	4
1932	26	2
1933	26	4
Total	78	10

Mortality: 12·8%.

TABLE III.

Ruptured Gastric Ulcer.

Year.	Total Cases.	Deaths.
1931	21	2
1932	36	6
1933	24	3
Total	81	11

Mortality: 13·5%.

TABLE IV.

Acute Obstruction of Small Intestine.

Year.	Total Cases.	Deaths.
1931	16	6
1932	14	8
1933	11	4
Total	41	18

Mortality: 44%.

TABLE V.

Strangulation by Bands.

Type.	Total.	Deaths.
1. Post-operative origin	9	5
2. Bands of unknown origin	21	9

The relative frequency of gastric and duodenal perforations, as shown by the records of the Adelaide Hospital, make one suspicious of the accuracy of the case records, but the total number of deaths in the series of perforations is indisputable, and is higher than it should be.

In the cases in these two groups the practitioner is called in to see a patient complaining of intense abdominal pain; and the patient's life will depend upon the practitioner's ability to answer correctly the question: "Is a laparotomy urgently necessary?" or: "Can I wait some hours for a reexamination?" These few hours are important, because one has to remember that after the practitioner has given his advice for immediate laparotomy a considerable and important period may elapse before the abdomen is opened, while the patient is being transported to hospital *et cetera*. Obviously every practitioner will make a tentative diagnosis, a detailed diagnosis being often impossible thus early; but one must be prepared to act on a tentative diagnosis, for the sake of early operation. It is unnecessary for me to emphasize the value of a searching inquiry into past illnesses, dyspepsia, and past operations.

The contribution that I wish to make to this evening's discussion is this: Under the two conditions of perforation of a viscus, and in internal strangulation, there are practically always two features present which are sufficient to determine the need for laparotomy. Both groups are characterized by the sudden onset of intense pain and by the presence of the most profound shock and collapse. In perforation of the viscus the pain tends to be persistent, whereas in strangulation, while slight pain may persist, it is obvious that the pain tends rapidly to assume the nature of a colic. First let us consider perforation of an abdominal viscus. The sequence of events is generally as follows: sudden onset of intense pain; profound shock and collapse; the pain is persistent; abdominal physical signs make their appearance, such as abdominal rigidity, and the presence of free fluid in the abdominal cavity, tympany *et cetera*.

The careful eliciting of the patient's history in most cases will enable one to determine which viscus has perforated; but the point that I wish to emphasize in this paper is the significance of the combination of that sudden onset of severe abdominal pain and extreme collapse. I believe there is only one extraabdominal catastrophe which may lead to confusion on this point, and that is the occurrence of coronary thrombosis. Most of us have seen the case of coronary thrombosis with intense epigastric pain of sudden onset, and the most profound collapse; but if the tachycardia and low blood pressure, and absence of any abdominal physical signs are borne in mind, the mistake of operating upon a case of coronary thrombosis will not be made.

Secondly, as regards internal strangulation, we have the following picture: sudden onset of intense abdominal pain; extreme shock; pain of a recurrent and colicky nature, with exacerbations of the signs of shock with each wave of pain; the absence of abdominal physical signs in the early and hopeful stage.

It is true that not all internal strangulations are characterized by intense pain, nor are they all associated with extreme shock; but I believe that if the two features of intense abdominal colic and profound collapse are found together in a patient, they justify a laparotomy without waiting for any of the classical signs of acute obstruction. The following case illustrates that point:

Mr. N., aged forty-four years, was lying in bed in hospital one afternoon during his convalescence from an upper abdominal operation which had been performed some twelve days beforehand. At 3.30 p.m. he was seized by sudden intense pain of a colicky nature and severe collapse. When seen about half an hour later he was

obviously suffering from intestinal colic, his colour was a pale ashen hue, his temperature was 35·1° C. (95° F.), his pulse rate 60, with low volume, his skin was cold, and he was perspiring profusely. He referred his pain constantly to the umbilical region. Within one and a half hours of the onset of his pain the abdomen was opened and it was found that he was suffering from a volvulus of the lower one-third of his small intestine; the abdominal cavity contained a surprisingly large amount of straw-coloured, slightly turbid fluid. The volvulus was reduced and he made an uninterrupted recovery.

These descriptions of the clinical appearance of such conditions obviously do not paint the picture completely in either case; the picture changes every hour with the production of fresh signs, but these are the signs of complications, not of the initial lesion. They are an endeavour to describe the initial signs—almost constantly present early—and they are the signs we most need to interpret.

Presence of Free Fluid in Strangulations.

I wish to draw attention to the significance of free fluid in the abdominal cavity in cases of external and internal strangulation. It is a common observation that in strangulated external hernia free fluid is to be found, not only within the sac, but also within the general peritoneal cavity. And it is often sufficient in quantity to be capable of pre-operative diagnosis in both situations. The explanation of blood-stained fluid within the hernial sac is obvious enough; but I am not certain of the explanation of the fact that the general peritoneal cavity so often contains a large quantity of clear, straw-coloured fluid. It must be a response of the whole mesenteric and omental peritoneum to a violent stimulation of a small localized portion of the mesenteric nerve plexuses. But in my limited experience the fluid has been greatest in quantity and most rapid in production in those cases in which a portion of the mesentery itself was strangulated and in danger of gangrene. It has been most in evidence in cases of internal strangulation; and I believe that, when found in a suspicious case, the presence of free fluid in the abdominal cavity indicates the need for early laparotomy. This sounds only too obvious; but recently, in one afternoon at the Adelaide Hospital, two patients with impacted hernia were admitted.

One had a femoral hernia of six hours' duration, the other a big inguinal hernia of many years' standing, which had been impacted for over twenty-four hours. Neither patient appeared to be in any distress, nor were any of the classical signs of acute obstruction present. There was some doubt in each instance as to whether the sac contained free fluid; there was no doubt, however, that free fluid was present in the abdominal cavity of each patient. Although each patient had suffered impaction frequently before, with manipulative reduction, the presence of free fluid determined immediate operation. In the femoral hernia a small loop of small intestine was dark purple, but was obviously viable. In the inguinal hernia, of longer duration, a much larger loop and a mass of omentum were almost black, and the viability was doubtful. Subsequent events justified the return of the bowel.

I wish to emphasize that, apart from the presence of ascites, neither patient gave the impression of being on the verge of gangrene of the small intestine.

Reviews.

CHRONIC NASAL SINUSITIS.

"CHRONIC NASAL SINUSITIS AND ITS RELATION TO GENERAL MEDICINE", by Patrick Watson Williams, is a book of outstanding value and importance.[1] The necessity of the close association of the general physician and the oto-rhinologist in the treatment of many general infectious disease is stressed.

The book demonstrates how intimately disease of the nasal sinuses is associated with general medicine, and how treatment of these infected cavities is essential to the more complete recovery of the patient.

Sir Humphry Davy Rolleston writes in a foreword: "This important work is an admirable example of the broad outlook which is so essential in the mental attitude of those who concentrate their attention on one part of the body." Again, it must appeal to the profession generally both from the point of view of accurate diagnosis of many otherwise obscure and different cases, and also because it has such a direct bearing on the ideal of medicine, namely the prevention of disease.

The author has divided the subject into three parts. In Part I he deals with pathology, symptoms, and systemic effects. After a general introduction, the pathogenesis of focal sepsis is fully described; diagnosis, symptoms and signs are then considered.

An interesting chapter on sinusitis in children is included. The author points out the possible very early onset, antral sinusitis occurring at two years of age, and ethmoiditis as early as six or eight months. The importance of correcting hypovitaminosis in these cases is stressed. The author states that chronic sinusitis should be suspected if removal of tonsils and adenoids is not followed by good health. Regional complications are thoroughly dealt with, including those involving the nervous, respiratory and joint systems *et cetera*.

Part II deals with diagnosis methods and treatment. The author advocates endo-rhinoscopy in preference to the ordinary indirect posterior rhinoscopy, and has included many drawings illustrating the pictures thus seen.

In agreement with many oto-rhinologists, he notes the unreliability of transillumination as a method of diagnosis of infected nasal sinuses, whereas skiagrams are one of the most valuable aids.

One of the most important parts of the book is the description of the author's own original methods of suction exploration and disinfection of the nasal sinuses. This method is recognized and practised throughout the world. The author's technique in operating on the sinuses either by the pernasal route or by the external route is described in a most practical way. Radical procedures are advocated only in very advanced cases of infection. Vaccine therapy in the treatment of chronic nasal sinusitis is regarded as of very doubtful value, unless the infected sinuses have been previously opened and drained. Non-specific protein therapy and ionization find very little to recommend them.

In Part III, under the heading of "Some Problems in Chronic Sepsis", agranulocytosis, mixed infections and obscure bone disease are discussed, and their relationship to chronic nasal sinusitis is stated.

Altogether this is a book of immense value, both to the general physician and to the oto-rhinologist, and is to be highly recommended. It is easy to read and understand, the author possessing that art of clear practical description which makes for the enjoyment in reading a book.

There are 262 pages of clear print, including a complete index, also some 122 fine illustrations and drawings. Many references to the literature on the subject are quoted, and these are conveniently placed at the end of each chapter to which they refer. The publishers are to be complimented on the beautiful little manual that they have produced.

[1] "Chronic Nasal Sinusitis and Its Relation to General Medicine (Chronic Sinusitis and Systemic Sepsis)", by P. Watson-Williams, with foreword by Sir Humphry D. Rolleston; Second Edition; 1933. Bristol: John Wright and Sons Limited. Royal 8vo., pp. 280, with illustrations. Price: 15s. net.

The Medical Journal of Australia

SATURDAY, JUNE 30, 1934.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

PASSING ANOTHER MILESTONE.

WITH this issue THE MEDICAL JOURNAL OF AUSTRALIA completes the twentieth year of its existence. The first number appeared on July 4, 1914, just a month before the declaration of war. Many will remember the series of events that led to the establishment of this journal—the feeling that closer union between the Australian Branches of the British Medical Association was necessary, the determination to bring that union about, the formation of the Federal Committee, the desire for a journal that would be the common mouthpiece of the Branches, and the arrival of the late Henry William Armit from England to take up the editorship. The name of Robert Henry Todd stands foremost among those who had the vision and the determination to carry into effect what they knew to be necessary. But the establishment of this journal was not the work of one man alone; the Branches in the six States brought to the project a spirit of cooperation, they sank their differences and joined hands in the common endeavour. Moreover, the New South Wales and Victorian Branches, by ceasing the publication of their respective journals, made possible the appearance of the new journal. It remained for Henry William Armit, with his journalistic training, his academic outlook and his driving force, to fashion of the new instrument a weapon, efficient, effective and durable.

During the first two decades of its life the journal has seen many changes. It could not have been started at a more difficult time. The World War caused a complete *bouleversement* of existing conditions, and those who controlled the journal were faced with economic problems of the greatest difficulty. The crisis passed, however, and in 1925 the Australasian Medical Publishing Company extended its activities; it erected its own building, purchased a complete printing plant and was able to print and publish the journal under its own roof and by its own staff. A second ordeal has had to be faced in the world-wide financial depression. From this ordeal THE MEDICAL JOURNAL OF AUSTRALIA and the Australasian Medical Publishing Company that controls it are emerging in a healthy state in which it may be claimed that the needs of the Branches and of their individual members are met.

What of the future? If the claims made for the journal be admitted, there is no justification for slackening of effort; still less, of course, is there any excuse for inactivity if these claims be not conceded. To be complacent about failure is pitiable; to be complacent with success is reprehensible. But it may be asked: "Why should the members of the British Medical Association in Australia be concerned in the future of the journal?" "Why should they feel any responsibility for its success?" The answer is obvious. THE MEDICAL JOURNAL OF AUSTRALIA belongs to the members of the several Branches of the Association—each member has a vested interest in it. The journal should portray the state of medical thought in the Commonwealth, and medical science cannot stand still. Occasionally complaints are heard that the journal is deficient in certain respects—the contributions are too scientific, or they are not scientific enough, there is not enough clinical matter, or sufficient space is not devoted to reports of medical meetings and so on. People who make these complaints are probably identical with those who find fault with meetings arranged by Branch councils or with measures initiated by

councils in the world of medical politics. The truth is that with the journal, as with the Branches, the member who would derive benefit must bring with him a helping hand and a receptive mind. Many would-be followers of Æsculapius are like the proverbial American tourist who "does" Rome in a day—they see and do not perceive, they hear and do not understand. It is imperative that during the next period of its existence THE MEDICAL JOURNAL OF AUSTRALIA should continue to make progress. Those directly concerned in its production cannot achieve this progress without the cooperation of the members of the Branches. Members can cooperate by reading the journal, by making their criticism constructive as well as destructive, by overcoming the lethargy characteristic of many clinicians, and contributing to its pages, and, finally, by taking part in the scientific meetings of the Branches and by helping to make the reports of these meetings worthy of the occasion.

Current Comment.

NOCTURNAL DYSPNŒA.

AN eminent teacher of medicine in Australia was accustomed to say that one of the tests of the able physician was the soundness of his opinion on the state of a patient's heart. Admitting that the eye of science cannot as yet make exact appraisal of the condition of the coronary arteries or even, in many cases, of the whole of the cardiac muscle, we must agree that the sound clinician should be able to guide his patient in the literally vital question of how much his heart can do. To do this he must weigh in the balances of his judgement the subjective and the objective evidence; on the one hand the accurately taken history must be assessed, on the other the findings of clinical and instrumental examination. All who practise medicine must become skilled in eliciting and evaluing the patient's own account of his feelings; therefore all the more interesting is a clinical study of the types of nocturnal dyspnœa by W. G. Harrison, junior, J. A. Calhoun, and R. T. Harrison.[1] This is one of a series of essays on various aspects of congestive heart failure, a general title which will serve to remind us of the lesion usually underlying the important and somewhat obscure attacks of nocturnal breathlessness.

Of the many symptoms of cardiac failure the two that head the list are pain and shortness of breath. The former may be regarded as Nature's red flag of danger, if not of revolt. The significance of pain in the treatment of cardiac failure, particularly in the ordering of the patient's life, is dealt with by Professor Carmalt Jones in the present issue of this journal. The latter is often more insidious in onset, but it occupies most of the patient's field of consciousness. In other words, he may be almost too breathless to be conscious of the lesser degrees of pain. Of course, the converse happens also, when the stricken man is seized by pain so urgent that his dyspnœa is mentally almost brushed aside. Among the more serious and severe types of breathlessness are the more or less sudden attacks of dyspnœa that occur by night, often waking the patient from sleep. Harrison, Calhoun and Harrison, in their article, first review the various theories that have been invoked to explain such seizures. The first they mention is that of back pressure, a term that is anathema to some clinical teachers, but nevertheless of certain rough descriptive value. Experimental and clinical evidence can be brought forward to support the hypothesis that there is a discrepancy between the relative outputs of right and left ventricles; in other words, that there is a left ventricular failure. Eppinger and others pointed out that diminution of the left ventricular reserve, together with increased venous return to the heart (due to peripheral vaso-dilatation) would produce an acute congestion of the lungs, thus causing dyspnœa. The mechanism involved in the lungs was thought to be reflex in nature. Eppinger and his co-workers also advanced an explanation for the occurrence of the dyspnœic attacks soon after the onset of sleep: they found that blood pressure and oxygen consumption reached their lowest levels within the first two hours of sleep, whereas the brain volume was increased during this period. Whether their argument of a decreased heart tonus associated with an increased venous return is sufficiently sustained by this evidence has been questioned. But it is interesting to note that a modern view of acute pulmonary œdema and nocturnal dyspnœa regards these as essentially the same, differing only in degree; this quite fits in with the theory just advanced. It must be pointed out, too, that this theory takes particular heed of the most striking feature of this singular symptom complex, the occurrence of the attack during a period of great apparent physical serenity. Harrison and his fellow authors mention a number of other hypotheses. There is, for example, that of insufficient aeration, sponsored by the famous Traube. This invoked the aid of pulmonary congestion, which probably none will deny; but that such a rigidity of the lungs (*Lungenstarre*) as Traube postulated could cause diminished aeration of the arterial blood has been disproved by many workers in this field. Acidosis has also been suggested, but there is no evidence that this can be a cause of nocturnal dyspnœa. Neither arterial blood nor the blood from the jugular vein has been found to show any appropriate alterations in hydrogen ion

[1] *Archives of Internal Medicine*, April, 1934.

concentration; therefore we cannot believe that an actual acid stimulation of the respiratory centre takes place. In fact, a mild alkalosis is not uncommonly present in the subjects of cardiac dyspnœa. The authors present also the views of those who advocate a nervous cause. A pulmonary reflex has already been referred to. One not unattractive idea is that of a disturbance of the water regulation of the body, causing a passage of fluid from the tissues into the blood stream during the night. Brunn, who made this suggestion, believed the patients suffering from nocturnal dyspnœic attacks to have latent œdema, and thought that a sufficient plethora might occur to cause pulmonary congestion. He supports his views with these facts: diuretics may prevent the occurrence of paroxysmal dyspnœa; large amounts of urine are frequently voided after attacks; patients suffering from cardiac failure often pass urine frequently at night; and, lastly, posterior pituitary extract aids in water retention by the tissues, possibly a reason for its value in the treatment of these night attacks. However, the authors remark that supporting evidence is rather lacking; they find, for example, that the hæmoglobin in such cases is actually less in the mornings than in the evenings, and that the oxygen capacity is higher. One of their clinical observations is very interesting in this connexion. Seven patients out of a total of thirty observed stated that they felt breathless when they desired to urinate, and that emptying the bladder was followed by relief of the dyspnœa.

This brings us to the results of the clinical study made of these thirty patients. Most of the patients were over forty years of age. The commonest underlying cardio-vascular lesion was hypertension and arteriosclerosis, next in importance came aortic regurgitation due to syphilis and lastly heart disease due to rheumatism. It was found that left ventricular strain was practically invariably present; that is, it was a common factor. Enlargement of the heart and lessening of the vital capacity were constantly found, and disturbances of rhythm were also frequently noted. Careful inquiry was made as to the exciting causes of the attacks, and the replies, when tabulated, give an excellent indication of the advice we should offer patients suffering from such disturbances. In order of frequency they were as follows: the position of the body (the recumbent posture was less comfortable than the sitting position), cough, the degree of activity during the preceding day, abdominal distension, large evening meals, constipation and desire for evacuation of the bowels, hunger, unpleasant dreams, undue heat, and a desire to urinate. It will be seen, then, that by advising a discreet life, attending to the digestive and excretory functions, preventing pulmonary irritation and securing adequate rest by proper posture in bed, and perhaps the use of sedatives, valuable help may be given these patients. Of course, this is not the whole tale of treatment, nor will it furnish the cure of these attacks, which are often urgent and not seldom unexpected; but we should remember that therapy does not concern drugs alone.

The authors distinguish several types of nocturnal dyspnœa. One is not truly paroxysmal; it develops slowly throughout the afternoon and becomes acute at bedtime; this they call "evening dyspnœa". Another variety is that occurring just as the patient falls asleep. He is repeatedly awakened by shortness of breath, but is able to rest throughout the night if he can but fall soundly asleep. This is probably a variant of paroxysmal Cheyne-Stokes breathing, and such patients seldom are attacked by acute pulmonary œdema. On the other hand, other patients tend to suffer from œdema of the lungs; in these the dyspnœic attack more frequently awakens the patient out of deep slumber. These types of night breathlessness may be mingled and appear in combination form in the same patient.

This study concerns only the types and causes, both underlying and exciting, of nocturnal dyspnœa. Treatment must firstly be directed towards the indications given by the cardiac disease observed in the individual patient, next to the relief of symptoms, and lastly, the prophylactic measures above suggested should be noted.

It is of great interest to contrast the picture of this syndrome with that of effort angina. In the latter undue effort invites its own penalty; in the former there is also effort involved, but this is visceral, not primarily somatic, and largely out of the control of the patient. Therefore neither the explanation of the phenomenon nor its relief is so easily gained. The limitations of cardiac failure are severe enough where they narrow the activities of the day, but when is added the terror that walks by night, life becomes a burden. Thus it is obvious that nocturnal dyspnœa, a symptom so serious and so distressing, calls for further studies in both the experimental and clinical fields.

Special Articles on Treatment.

(Contributed by request.)

XXXIV.

THE TREATMENT OF THE FAILING HEART.

THE function of the heart being to maintain the circulation of the blood, heart failure means that the heart is not contracting with adequate force. Such failure may be intrinsic or extrinsic in origin, that is, the heart may be intact or nearly intact itself, but may suffer from the structural or functional disorder of other systems, or from the deficiency of essentials from the blood, or from the presence of poisons. In all such cases treatment must be directed to the organ or system chiefly at fault, if the principle of removal of the cause is to be observed; just as it is necessary first to treat the heart for dyspepsia due to venous congestion.

If, therefore, one takes dyspnœa, tachycardia, palpitation and precordial pain as the common obtrusive symptoms of cardiac failure, it is necessary to be sure that the case is not primarily one of chronic bronchitis, phthisis, renal disease, arterial hypertension, hyperthyreoidism, anæmia or diabetes, before undertaking treatment aimed at the heart.

Such treatment is reserved for cases of intrinsic heart failure, whether the cardiac defect is due to rheumatism, syphilis, or coronary disease. In every case, what happens is failure on the part of the heart muscle to discharge its functions. Those functions are multiple and one or more of them may fail in any case, and treatment is more efficient with some than with others. The functions of the heart muscle as summarised by Mackenzie[(1)] are stimulus-production, excitability, conductivity, contractility and tonicity.

Contractility, which gives the necessary force to propel the blood, is in itself the most important of these, but efficiency depends on the proper coordination of contractions which is secured by stimulus-production, excitability and conductivity. Tonicity is a function of great clinical importance, because the retention of tone in a case of heart failure leads to anginal pain, and relaxation of tone to congestion.

Heart Failure with Angina.

The work of recent years has made an important subdivision of cases of heart failure with angina; and "angina of effort", Heberden's angina, is separated from the anginal pain of coronary thrombosis. There is close association between them. A patient who has survived an attack of coronary thrombosis is fairly certain to suffer from angina of effort thereafter; and also a person who suffers from angina of effort is a likely subject for coronary thrombosis; but the two conditions, angina of effort and coronary thrombosis, are profoundly different and must be treated independently.

Those of us who in our youth found inspiration in the work of Sir James Mackenzie[(2)] were satisfied that the viscera are insensitive and that visceral pain is felt in the body wall and is due to the contraction of a hollow viscus against a resistance which it cannot readily overcome, though that precise phrase may not occur in Mackenzie's writings. Hurst[(3)] modified this by assuming that the viscera are sensitive to tension, and that this is the only cause of true visceral pain. These investigators were dealing at the time with the alimentary tract; but the heart is a hollow viscus and the same considerations apply to it. Lewis[(4)] states that muscular ischæmia, defective blood supply, absolute or relative, is the cause of anginal pain, and he postulates a specific chemical or physico-chemical change in the musculature of the heart. However that may be, muscular ischæmia will certainly add to the difficulty of a hollow viscus in overcoming resistance to its contraction. If, then, cardiac pain, however induced, is due to defective blood supply relative to the work required of the heart, the first and most obvious step in its treatment is rest. The pain varies from discomfort to agony. It is substernal in situation and is, according to Mackenzie, over the distribution of the upper four left dorsal nerves, sometimes reaching those of the seventh cervical and the sixth dorsal and occasionally bilateral. Mackenzie used also to insist on a sense of constriction of the chest and of a feeling of impending dissolution, the latter is certainly not invariably present. If, as Lewis states, the pain is due to muscular ischæmia, it is naturally continuous in coronary thrombosis when the ischæmia is permanent, while it varies with exercise in angina of effort.

Coronary Thrombosis.

The commonest vessel in the heart to be occluded is said to be the descending branch of the left coronary artery, and the result of occlusion is of course that a part of the left ventricle is put out of action. A brief account of a case recently studied will illustrate the problem and its treatment.

A medical man, aged 63 years, a heavy smoker and inhaler, but almost a teetotaller, and previously in good health, began to suffer from undue fatigue, dyspnœa on exertion and precordial pain, of no very great severity, but alarming from his knowledge of its implications. On one occasion, when out motor driving, he felt so ill that he thought it wise to stop his car, lest he should lose control of it on a hill. He walked about a hundred yards with great difficulty to a friend's house, and collapsed in the hall. He was in severe distress and considerable precordial pain and his pulse was feeble and irregular; he was given injections of morphine and removed to his home in an ambulance. The diagnosis of coronary thrombosis was made. Next day the patient was quite rational, the condition was discussed with him, and it was agreed that he should take six weeks' complete rest in bed.

The symptoms in this case were the sense of collapse, extreme asthenia, and precordial pain of considerable but not intense severity. There was persistent vomiting and the patient took no food for four days; after this the vomiting ceased, the appetite was restored, and the bowels gave no trouble. There was severe aching pain in the limbs. The temperature was irregular, and rose as high as 38·9° C. (102° F.) for ten days. Sleep was very difficult to obtain, and morphine was required, 0·015 gramme (a quarter of a grain) at 8 p.m., and 0·01 gramme (one sixth of a grain) at midnight. Pericardial friction soon appeared and continued for some days.

Treatment: The patient was kept at very complete rest in bed, morphine was given regularly in the doses mentioned, and light diet was given to suit the patient's inclinations. After four weeks, massage of the limbs was started at the patient's request, which proved rather exhausting and increased the muscular pain. It is not advised.

The pulse rate was low, but regular, occasional extra systoles occurred; the blood pressure was low, 100 millimetres of mercury, and this was considered the unfavourable point in the case.

After six weeks in bed the patient was removed to a seaside cottage where he stayed for a further six weeks, at first spending the day in a lounge chair in or out of doors, according to the weather. He very gradually began to take exercise and at the end of the second six weeks could walk half a mile without distress. He was then obliged, on financial grounds, to recommence practice. He found this extremely exhausting for about another three months, but by taking great care to avoid all unnecessary effort, he suffered no further breakdown. He gave up tobacco for six months, but has since resumed smoking to a moderate degree; this may induce slight precordial discomfort, and he is disposed to advise against it. It is now a year since the original attack, and his health is to all appearance better than it was before the thrombosis occurred; a contributory element to this may be the removal of his teeth on account of some degree of chronic *pyorrhœa alveolaris*. He considers himself quite free from symptoms; he proceeds with great caution, but is able to discharge all the duties of a medical man of senior standing, together with the instruction of classes of students.

This result was probably the most favourable that could be looked for, the treatment was merely that suggested by commonsense, and in it the patient fully cooperated. Apart from relief of symptoms, the treatment consisted in complete rest for a period long enough to permit the heart muscle, damaged by thrombosis, to be replaced by scar-tissue; this period is estimated by Lewis at eight weeks. At this stage no further benefit is to be looked for from rest, and the proper course is very gradual training of the remaining muscle, all exercise being kept strictly within the limits of fatigue. Tobacco was limited; it should be given up altogether. Focal sepsis was treated; this, however, requires very great discretion; I have known more than one patient killed by over-zealous dental extraction.

All cases of coronary thrombosis should be treated in a similar way, symptoms in other systems being treated as required. In more excitable people bromides or hypnotics may be necessary; none is specific. Morphine is essential for the pain, and, in contrast to the treatment of angina of effort, vaso-dilators should not be given to relieve it; in the first place they are useless and in the second they may be actually harmful, as they lower the blood pressure which is already dangerously low. Vaso-dilators may be used cautiously in the angina of effort to which patients

with healed lesions are subject; their use is discussed under the heading "Angina of Effort".

This case also suggests a word on prophylaxis; the patient had long followed a sedentary occupation but retained very good physical strength. At the age of 60 he undertook some very laborious and unaccustomed work in his garden which involved carrying a large number of heavy stones; this work occupied his leisure for several weeks. He is disposed to date his cardiac failure from this time. Most authorities are satisfied with Lewis's statement that it is impossible to strain a healthy heart by exercise, but a heart in which the vessels are undergoing early senile sclerosis is not precisely "healthy". Such work should not be undertaken in late middle life by persons unaccustomed to it; this case suggests that it may predispose to coronary thrombosis.

Such a result as that just described is only to be looked for in a lesion of limited extent, but the limit is impossible to guess and is quite out of the reach of treatment. The following case may be quoted in contrast:

A dentist of 45 years was accustomed to take a walk in the morning before his day's work. On the last occasion he felt suddenly ill while walking, was obliged to sit down, and had to be taken home in a car. He complained of intense precordial pain, radiating down the left arm; he was put to bed and morphine was injected. He was seen next day in consultation, some thirty hours after the "stroke"; he was then cold and sweating and frequently lapsed into unconsciousness; the heart-rhythm was slow and much broken, and a pericardial rub was present. The patient was clearly moribund, and died about twenty-four hours later. In this case the thrombosis was presumably ingravescent. The patient was treated on precisely the same lines as the former patient but could make no response.

Angina of Effort.

A patient who has recovered from an attack of coronary thrombosis, and is thus deprived of a portion of his cardiac muscle, is likely to suffer from cardiac pain whenever he makes any muscular effort, but such pain subsides as soon as he rests; it has not the persistence which characterizes coronary occlusion. A person whose myocardium is defective for any other reason, may suffer in the same way; recognized causes are senile change, probably associated with arteriosclerosis, high blood pressure, aortic disease, cardiac syphilis, and occasionally diphtheria; in fact any kind of heart lesion which is not associated with congestion. Lewis has described a type associated with high blood pressure and high pulse rate in cases of free aortic regurgitation. This study threw much light on the use of amyl nitrite in angina.

Angina pectoris (angina of effort) was described by Heberden as a painful and most disagreeable sensation in the breast on walking; as soon as the patients stand still the pain vanishes. It has recently been studied by Wayne and Laplace[5] at University College Hospital; these observers paid particular attention to the action of nitrites. The angina of effort is induced by slight exertion, need never be severe, and is stopped at once by rest; it is the form of cardiac pain commonly complained of. The symptom is generally observed in men in later life. A mild degree of it is frequently the first indication of heart failure with advancing years in New Zealand farmers and similar workers; doubtless the same is true in Australia. Occasional extra systoles are the only things then found on physical examination. Wayne and Laplace, at University College Hospital, studied some 400 attacks induced in eleven patients by exercise. The amount of exercise required to induce the pain was found to be very constant for each patient; the blood pressure was nearly always raised to a much greater extent by the amount of exercise taken than is the case with young persons after similar efforts, but the rise was variable and pain appeared at different blood pressure levels, conflicting with the commonly held opinion that a rise in blood pressure is the cause of cardiac pain and that its relief by nitrites depends on vaso-dilatation. On the other hand, a rise in pulse rate was much more closely associated with pain, and a fall of rate below a fairly constant level coincided with its relief. Similarly the pain was made worse if the pulse rate was quickened by atropine, and the pain was lessened by pressure on the carotid sinus, slowing the pulse. These writers quote authorities to the effect that the pulse rate is a better indication of the "internal work or energy expenditure" of the heart than the blood pressure. A high pulse rate indicates great energy expenditure, and that in these patients is associated with angina.

The use of the nitrites was fully investigated in this series. It was found that generally pain was immediately relieved by inhalation of amyl nitrite, but the duration of true angina of effort is so short, that rest is generally sufficient to relieve it, and many patients prefer not to undergo the unpleasant sensations induced by the drug. The nitrites reduce blood pressure but increase pulse rate. As these authors think, the latter is sufficient to counterbalance the former and they attribute the well known clinical efficacy of the nitrites to their dilating effect upon the coronary arteries, and to the consequently improved blood supply to the cardiac muscle. That a very real effect is produced on the heart muscle is shown by electro-cardiography. In cases of angina there is frequently a reversal of the *T* wave in one or other lead, which is attributed to some kind of defect in the cardiac muscle; the exhibition of nitrites restores the *T* wave to normal for a time.[5]

Treatment of Angina of Effort.

Since true angina of effort is excited only by effort, limitation of effort is the obvious first essential of treatment. When the pain comes on, it will be relieved in a few minutes by rest. Occasionally people are found in whom the pain subsides if effort is continued, but the reverse is usually the case. If it is desired to hasten the cessation of pain, amyl nitrite, 0·3 cubic centimetre (five minims) inhaled from a crushed capsule nearly always stops it; rarely the pain is not relieved and occasionally it is made worse. Patients using amyl nitrite for the first time should be warned to use it only while sitting or lying, and to start with the capsule held at arm's length and gradually brought up to the nose, otherwise it may produce sufficient dizziness to cause a fall. Nitro-glycerine acts more slowly; if given in doses of 1·3 milligrammes (one-fiftieth of a grain) more exercise can generally be taken without pain. Fresh *liquor trinitrini* is the best preparation; the tablets readily deteriorate. Wayne and Laplace found little value in erythrol tetra-nitrate.

In regard to prophylaxis, these observers found no advantage in keeping patients in bed; they were just as susceptible to pain when exercise was resumed. Evans and Hoyle[6] have made a most elaborate study of ninety patients over a period of two and a half years, in search of a drug or drugs which would reduce the frequency and severity of anginal attacks in the susceptible. Of the thirteen drugs which they employed they report: "We have been unable to convince ourselves that any drug tested is worthy even of a trial in the routine treatment of the disease".

In the prophylaxis of attacks of angina of effort, it has to be remembered that the pain may also be induced by the following: (*a*) Mental excitement, of which the case of John Hunter remains the classical example. (*b*) Cold. Exercise in a cold wind is highly provocative; going to bed in a cold room and getting between cold sheets should be avoided. (*c*) Taking food, and that not necessarily in association with dyspepsia. (*d*) Sleeplessness. Ammonium bromide is valuable in such cases. Naturally any infection, influenza, for instance, will precipitate attacks and should be guarded against with all care.

What advice should be given as regards exercise to patients who are subject to angina of effort? They should certainly not be made invalids and should be encouraged to take as much exercise as possible within the limits of fatigue and the induction of pain, and to realise that even a defective heart muscle is capable of training.

Other Varieties of Angina Pectoris.

Angina in Aortic Disease.

Lewis[60] has recently separated out a variety of angina with severe and long-continued pain which occurs chiefly in men who are the subjects of grave aortic disease. The attacks are associated with great increase both in blood pressure and pulse rate; they occur independently of effort, sometimes at night, and may be induced by food. They are promptly relieved by inhalation of amyl nitrite, and it is suggested that it is on such cases that the great reputation of this drug depends. The drug reduces both pulse rate and blood pressure, but not sufficiently to account for the relief of pain. Lewis infers that the attacks are due to a generalized vasomotor spasm, in which the coronary arteries are included; the relief given by amyl nitrite depends upon its vaso-dilator effect upon the latter. The patients are generally cardiac cripples whose condition is very grave, they are frequently bed-ridden, and amyl nitrite is essential to them for its symptomatic value; they should always carry capsules of the drug.

Secondary Angina.

Sir James Mackenzie[59] devoted considerable space in his book on *angina pectoris* to "secondary angina", which, not being really heart failure, need not be considered here in detail. Its diagnosis is, however, important; it is far commoner in women than in men, while the reverse is true of primary angina; exhaustion is a commoner complaint than pain. Patients with true angina are generally well between attacks, those with secondary angina are invalids. Chronic infections and psychical disturbances are often involved; Mackenzie regarded ammonium bromide as a great stand-by in their treatment. The essential treatment is that of the primary condition.

Angina from Allergy.

Persons who are subject to the commoner allergic symptoms such as asthma and migraine occasionally complain of quite typical *angina pectoris*. The cases are not sufficiently numerous for a large experience to be available, but the diagnosis is reasonable in allergic patients who show no other sign of heart lesion. The pain probably results from spasm of the coronary arteries and can be relieved by adrenaline, 0·3 cubic centimetre (five minims), injected 0·06 cubic centimetre (one minim) at a time, as for asthma. Desensitization is probably a wise course in such cases; subcutaneous injection of a 5% solution of peptone is the most accessible method, a suitable dose is one cubic centimetre weekly for a month, followed by the same dose at longer intervals for another two months.[60]

Tobacco Angina.

There is a tradition that tobacco induces angina. Mackenzie did not discuss it in his books and Lewis mentions it only in passing. I have certainly seen one case in a middle-aged heavy smoker of very severe pain of anginal type which the patient found to be associated with smoking, and which disappeared when he gave up the habit. Observant patients who have survived coronary thrombosis seem satisfied that tobacco is injurious to them. It should be forbidden to patients suffering from *angina pectoris* of any origin.

The Gall-Bladder and Cardiac Pain.

John Hunter had gall-stones as well as coronary disease, a fact emphasized by Mackenzie. C. H. Miller[61] has noted a close resemblance between the pain in the two different conditions, and quotes several cases of cardiac pain abolished by surgical treatment of the gall-bladder. The association should be borne in mind; that is, the two conditions may co-exist, and also pain like angina may be induced by disease of the gall-bladder, and cured by its treatment.

Heart Failure with Congestion.

It sometimes happens that a patient subject to angina ceases to have pain, but becomes cyanosed and œdematous. The clinical inference is that the tonicity of his heart muscle has given out and that the heart or part of it is dilated; but I am not aware that there has been any experimental demonstration of this. In many cases of heart failure there is congestion from the beginning, particularly in those of rheumatic origin. In congestive heart failure there is an important division between hearts with regular rhythm and those with broken rhythm, or, what is more to the purpose, between cases of auricular fibrillation and all others.

Auricular Fibrillation.

Lewis[62] has stated that between 60% and 70% of all patients with heart failure admitted to a general hospital show auricular fibrillation; its importance is therefore manifest. Heart failure with auricular fibrillation is recognized clinically by signs of congestion with rapid and completely irregular heart beat, some of the beats failing to reach the wrist, so that the heart rate at the apex is greater than the pulse rate in the radial artery. Its clinical interest lies in the fact that it is peculiarly susceptible to treatment by digitalis and allied drugs. The condition is well known to be one in which the auricle fails to make effective contractions, attributed by Lewis to a "circus movement" of the wave of contraction through it, the result being that irregular stimuli from the auricle reach the A.V. node and a rapid and irregular ventricular beat results. The rapidity may be very great, the recovery period is inadequate and the ventricular beat loses force, with consequent circulatory failure. It is rarely possible to stop the fibrillation in the auricle; but the symptoms are relieved if the ventricle can be slowed, when it automatically improves in force. Digitalis effects[63] this by two different kinds of action; it inhibits the whole heart by stimulation of the vagus, and it also interferes with conduction along the bundle of His. Digitalis, then, is of use in auricular fibrillation because it inhibits the heart, and because it impairs conduction; it does not change the fibrillation in the auricle, and as it only partly inhibits the heart and impairs but does not destroy conduction, so the heart beat, though slowed, remains irregular. It is said to be possible to abolish conduction and induce a slow regular ventricular beat, but the drug is not pushed to this extent in therapeutics.

The patient with cardiac failure with congestion, therefore, upon whatever physiological system the incidence of the symptoms chiefly falls, is put to bed, and his bed is so arranged that he can sit vertically upright if need be, and if possible a bed-table is arranged across his knees so that he can rest his elbows on it and support his head on his hands. Some patients do better sitting up in an arm-chair. The diet should consist of small dry meals and the fluid should not exceed a pint a day. Lewis advises 800 calories a day for any period up to a week; the appetite is generally poor. It is important to avoid anything that may give rise to flatulence.

The drug treatment is by digitalis. For the present method of using this old-established drug we are indebted to Mackenzie, who was the first to recognize its specific value in auricular fibrillation. He described his method with remarkable clarity:[64]

> The best way, in cases of marked failure, is steadily to push the drug . . . until a reaction is observed. Usually the digestive system is the first affected, loss of appetite, nausea, vomiting or diarrhœa being set up, the patient usually feeling ill and miserable. If the digitalis is effective on the heart, as a rule a marked slowing of the pulse is found. . . . When this stage is reached I always stop the administration of the drug for a few days. . . . The heart rate is carefully observed, and when the rate shows signs of increasing, half doses of the drug should be given, . . . increased or diminished according to the manner in which it affects the rate. A good deal of my work has been done with the tincture of digitalis, and I may say that I have used this preparation for over thirty years and have never yet come across an ineffective preparation. . . . I start with 1 drachm of the tincture per day in doses of 15 to 20 minims. . . . Usually a reaction is obtained within a week.

The only other preparation of which he approved was Nativelle's granules of digitalin, one granule, 0·27 milligramme or one-two hundred and fortieth of a grain being equivalent to 0·9 cubic centimetre (15 minims) of the tincture. Mackenzie was emphatic on the danger of pressing the drug after it has produced its effect, since sudden death occasionally occurs.

It is usual to stop administering the drug when the pulse rate has reached about 80 per minute and to resume it when the rate begins to rise, proceeding by trial and error till a dose is found which maintains a rate of about 80 per minute at rest. Intelligent patients soon manage to regulate the dose for themselves. The most valuable objective indication of improvement with digitalis is increase in the amount of urine passed.

This method of administration is probably the most popular in British medicine today; there are perhaps only three modifications of importance. Of late years a standardized preparation of the leaf, *digitalis folia*, has been introduced which is often tolerated when the tincture induces vomiting, one grain of the leaf is equivalent to 0·6 cubic centimetre (10 minims) of the tincture. Physicians who have once used it generally prefer it to the tincture; it has the additional advantage of small bulk and portability, which is of great value to chronic patients who require to have the drug at hand. Quite recently E. J. Wayne[114] has worked out the therapeutics of digoxin, which is a pure and stable glucoside of the digitalis group, and requires no biological standardization as do the ordinary preparations of digitalis; it can be given intravenously in dose of 0·75 to 1 milligramme or 1 to 1·5 milligrammes by mouth. It has not yet come into everyday therapeutics.

Mackenzie's method takes about a week to give relief. In severe cases, if this period can be safely shortened, it will be of great advantage; this can be done by the method of Eggleston.[113] Eggleston found that in animals the amount of digitalis required to produce toxic symptoms was proportional to their weights and that therapeutic results require almost toxic doses; he worked out the dose for man and expressed it as a formula of "cat units per pound". The drug is absorbed in six hours, so the doses are spaced accordingly, and no toxic symptoms due to a given dose will come on after that time. Fraser[115] in an early report used digitalis on 14 severe cases with 11 good results, giving 5·25 cubic centimetres, 3·5 cubic centimetres and 1·75 cubic centimetres (1·5, 1·0 and 0·5 drachms) of the tincture at intervals of six hours. Elsie Porter,[116] from the Ancoats Hospital, Manchester, advises a "massive dose of the tincture of digitalis at the rate of 0·125 cubic centimetre per lb. of body weight, administered by mouth in a single draught" or, in cases of vomiting, a similar dose *per rectum* at the rate of 0·1 cubic centimetre per 0·45 kilogram (one pound) of body weight. Lewis, in his recent book, without reference to Eggleston, advises 5·25 cubic centimetres to 7 cubic centimetres (one and a half to two drachms) for an adult of average weight, to be repeated next day if required, with smaller doses subsequently, but he very clearly indicates that such doses should be used only on patients under close observation and by experienced practitioners.

The safe rule is to stop using digitalis as soon as any gastro-intestinal disturbance is felt, nausea, vomiting, or diarrhœa; only a very experienced person can select the case in which it should be continued. This vomiting after digitalis is of central origin; an untreated patient with gastric symptoms due to congestion may often have them relieved by digitalis.

In patients who are gravely distressed when first seen, or much troubled with vomiting, strophanthin, 0·26 milligramme (one-two hundred and fiftieth of a grain), is rapidly effective. It must, however, be given intravenously and should not be given along with digitalis. It may be repeated in six to 12 hours, but not sooner. In Manchester the single heroic dose of 0·001 gramme (one-sixty-third of a grain) is advised (Porter).

It is only in cases of paroxysmal fibrillation that a normal rhythm is ever likely to be restored after digitalis; in the great majority the rhythm remains broken. In recent cases which have responded well to digitalis, regularity can sometimes be restored by the use of quinidine, and, if so, the efficiency of the heart is a good deal improved. The cases to be selected[30] are those with symptoms of recent onset and short duration, with small hearts, and in middle age rather than youth. Those to be avoided are actively progressive cases—cases of acute Graves's disease, those not responsive to digitalis, and those of aortic incompetence and heart block. Clark Kennedy gives 0·39 gramme (six grains) every six hours on the first day and increases the dose by 0·065 gramme (one grain) daily (doses of 0·455 gramme (seven grains) on the second day and of 0·52 gramme (eight grains) on the third, and so on) till the pulse steadies or toxic symptoms arise, such as headache, nausea, vomiting, and giddiness. He found normal rhythm was restored in 80% of 45 cases, but it may be maintained for only a few days or for as long as a year; a maintenance dose has to be found in the successful cases. I have used quinidine in a number of cases, but have had much less frequent success, with almost invariable relapses. For this reason I now rarely prescribe it, in spite of one particularly favourable case in a student who started with auricular flutter; this was converted to fibrillation by digitalis and reduced in frequency, and then rendered permanently regular by quinidine. This patient afterwards "put the weight" for his university. Quinidine has perhaps a special value in the heart failure with auricular fibrillation which accompanies the crises of toxic goitre. Such thyreotoxic auricular fibrillation is associated with an active toxic cause, removal of which by partial thyreoidectomy generally restores sinus rhythm; if not, rhythm is often restored by quinidine.[30]

In thyreoid cases many observers think digitalis and quinidine useless and give iodine as Lugol's solution. Plummer,[30] who is a great advocate of iodine therapy, gives from 0·6 to 6 cubic centimetres (10 to 100 minims) of Lugol's solution three times a day in crises. As seen in New Zealand, this treatment is often absolutely useless. However, the condition is always a very grave one, few drugs are of the smallest use, and the iodine is worth a trial. Sedatives, the bromides, luminal, or hyoscine in the usual doses, are probably more reliable.

Heart Failure with Normal Rhythm.

Parkinson and Clark Kennedy[30] have studied the question of heart failure with normal rhythm. Rhythm is not disturbed in heart failure caused by acute infections, and may remain normal in cases of gradually developed obstruction of the circulation, systemic or pulmonary, and in gross lesions of the coronary arteries. The prognosis is less favourable than in cases of auricular fibrillation. As to treatment, all these authors have to say is: "For heart failure with auricular fibrillation we have at our disposal either digitalis or quinidine. Apart from syphilis, for heart failure with normal rhythm, we have no special treatment. Digitalis does benefit some of these patients to a large extent."

Hay, Wallace Jones and Ince[30] state that the use of digitalis is indicated in cardiac failure with normal rhythm when œdema is present, and that no other definite indication for the use of the drug has been found in their series, though clinical improvement may occur in some cases with œdema. This must surely be everyone's experience. There is, in fact, no other drug to use in cases of heart failure with congestion, and sometimes relief follows its use in the most unlikely cases. This is the doctrine of so distinguished a clinician as Wenckebach:[30] "Digitalis is indicated in all cases of heart failure, where insufficient functioning of the heart is the cause of the pathological condition. This holds good irrespective of the cause of the heart failure itself," and he will have nothing to do with the frequently cited exceptions of arterial hypertension, aortic valvular disease, aortitis or slow pulse.

Adjuvant Treatment.

Digitalis and quinidine are aimed directly at the heart, but in grave cases of heart failure with œdema and congested lungs, the heart works at a great disadvantage and accessory treatment is beneficial and even essential.

Immediate relief of distress in a cyanosed patient with overfilled veins in the neck and enlarged liver may be given by bleeding, the rapid withdrawal of 300 to 600 cubic centimetres (10 to 20 ounces) of blood from a vein in the arm. This may be done by simple incision of, say, the median basilic vein over a basin, but the resulting mess may be avoided by the use of French's apparatus, a large-bore tapered needle connected by tubing to a glass bottle, in which a partial vacuum is produced by a suction bulb. Leeches seem to have passed out of fashion; but the application over the liver of six leeches, which only remove an ounce or two of blood between them, gives sometimes remarkable subjective relief.

Dyspnœa may also be relieved by oxygen, best given by nasal catheter passed into the pharynx and secured to the face by strapping. The oxygen is passed from the cylinder through a bottle of warm water; a just continuous stream of bubbles is maintained.

Congested lungs may often be relieved by the use of large doses of ammonium carbonate, 1·2 grammes (20 grains) with 4 cubic centimetres (1 drachm) of syrup in 30 cubic centimetres (one ounce) of water. Three doses may be given at four-hourly intervals. When successful this measure leads to very profuse expectoration and considerable relief. In patients kept awake by cough, a dose at bedtime will often clear the lungs and permit sleep; if that is not sufficient it is best to use powerful sedatives. The following is a good prescription:

℞

Morphinæ hydrochloridi 0·03 gramme (one-half grain).

Apomorphinæ hydrochloridi 0·03 gramme (one-half grain).

Heroinæ hydrochloridi 0·03 gramme (one-half grain).

Acidi hydrochloridi diluti 4 cubic centimetres (1 drachm).

Syrupi pruni virginianæ 24 cubic centimetres (6 drachms).

Aquam menthæ piperitæ ad 180 cubic centimetres (6 ounces).

Signendum 15 cubic centimetres (half an ounce) *si opus sit pro tussi.*

The ordinary sedatives, chloral and potassium bromide, "Luminal", 0·03 to 0·06 gramme (one-half to 1 grain), and, if necessary, morphine, 0·01 to 0·015 gramme (one-sixth to one-quarter grain) hypodermically, may be given as required. Paraldehyde, unpleasant as it is, is a most valuable hypnotic, it may be given by the rectum, 8·0 to 16·0 cubic centimetres (two to four drachms) in 120 cubic centimetres (four ounces) of olive oil.

Fluid in the pleural cavity or the abdomen should be removed by aspiration if sufficient to cause embarrassment. Œdema of the extremities may be relieved by Southey's tubes or by simple incision, but the former method is unsatisfactory from the point of view of asepsis and the latter makes a horrible mess; sterile dressings are, of course, required. It is best to dispose of the fluid by the kidneys if possible. Polyuria indicates that digitalis has been successful, but if that action is delayed, "Diuretin", a derivative of theobromine, may be given in doses of 0·6 to 1·8 grammes (10 to 30 grains) three times a day. It is often not very successful, and my own preference is for salyrgan, which contains 35·5% of mercury. Ammonium chloride 1·8 grammes (30 grains) is given three times a day for three days before the "Salyrgan", and throughout the period of use of the latter. "Salyrgan" is put up in capsules, and 0·5 cubic centimetre is injected into the buttock muscles, and if there are no bad symptoms the dose is increased by 0·5 cubic centimetre every three days, till 2 cubic centimetres are given, and the injections stopped. This dose should not be exceeded. A more rapid result is obtained by intravenous injection, but the drug causes necrosis if it leaks into the subcutaneous tissues; it should be diluted in 10 cubic centimetres of normal saline solution and injected very slowly into the vein. In successful cases it leads to very profuse diuresis and relief of œdema.

There are cases of cardiac œdema which respond particularly well to the use of vitamin *B*, most readily given as marmite, 12 grammes (3 drachms) three times a day. The most suitable patients are those with rather soft œdema present in both arms and legs. The treatment was suggested by Wenckebach's description of the beri-beri heart, in which there is extensive œdema which resists cardiac drugs but responds to vitamin *B*.[26][27]

It may be worth while to call attention to the variety of systems upon which heart failure may fall, sometimes misleading the clinician in his diagnosis. I have known three patients with cardiac failure sent into hospital with a diagnosis of gastric ulcer on account of hæmatemesis; also one with melæna and one diagnosed as having parenchymatous nephritis. Eczema of the extremities is another occasional indication of heart failure in the susceptible.

On the other hand, failure of the right side of the heart may be induced in cases of severe chronic bronchitis and emphysema, and may be relieved by venesection and ammonium carbonate. If something can be done for the lungs the condition will right itself. Such patients, however, are occasionally rather grey than blue, and then suggest the condition to which W. T. Ritchie[28] has drawn attention as "acute circulatory failure", and as occurring in patients with pneumonia who collapse. He ascribes it to failure at the periphery and considers that it is due to exhaustion of the vasomotor centres and damage to the capillary walls; the blood pressure is very low. I have found this condition in a chronic bronchitic and have given great relief with adrenaline and ephedrine.

THE LIABILITY TO HEART FAILURE.

A word may be said in conclusion on the risk of heart failure among persons suffering from rheumatic heart disease. Poynton has remarked of a series of patients at the Hospital for Sick Children, Great Ormond Street, that 66% left with organic heart disease, and 22% were invalids. All observers are agreed on the need for prolonged convalescence after acute rheumatism, for the selection of suitable occupation and remedial recreation. On the other hand, it is most important not to make invalids.

In New Zealand (and probably in Australia) acute rheumatism is a much less urgent problem than in England. Still, a fair number of cases occur and I have been frequently struck with the long period of useful life which many persons have enjoyed before a breakdown. The following patients have been in Dunedin Hospital, out of many with recent heart failure:

1. A shearer, aged 60, who had a cupping-mark on his breast, the result of treatment given on account of a heart murmur when he was in the hospital as a boy with a broken arm. Heart failure was recent.

2. A man, aged 40, who served throughout the War as a farrier. His heart failure was recent.

3. A man of 36, a mechanical engineer, who was rejected for a lodge at the age of 21 on account of his heart. His heart failure was recent.

4. A man of 24, a farm-worker, with recent heart failure; he had had rheumatic fever ten years before.

These men had led perfectly normal, vigorous and useful lives for periods between ten and fifty years, in spite of heart lesions. Surely they have had more out of life than if they had been advised to live as semi-invalids, with the added neurosis which generally attends such cases. Let convalescence be long by all means; but once the patients are fit to resume ordinary life, they should be exercised to the full within their limits of fatigue. Breakdown may indeed come, but it may be so long delayed that one feels justified in this advice being invariably given to students: "Never make invalids".

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British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Anatomy Lecture Theatre, University of Adelaide, on February 22, 1934, DR. E. BRITTEN JONES, the President, in the chair.

The Diagnosis of Acute Conditions of the Abdomen.

DR. L. C. LINDON read a paper entitled: "Some Remarks on the Diagnosis of Acute Conditions of the Abdomen" (see page 836).

DR. BRONTE SMEATON congratulated Dr. Lindon on his success in gathering from an ocean of experiences some valuable pearls of wisdom. He thought that more importance might be directed to the suddenness of the onset in cases of rupture of the hollow viscera; the effect was comparable to that following a bullet wound. Diagnosis of such accidents as rupture of a viscus, internal hernia *et cetera*, was a simpler problem shortly after the occurrence, but became more difficult as time went on. He concurred with the counsel of Dr. Lindon that exploration should solve doubtful cases, and, looking back on a long vista of cases, considered that indecision and delay had been the outstanding error.

DR. G. H. BURNELL wished to congratulate Dr. Lindon on the excellent manner in which he had presented his case for early diagnosis. Dr. Lindon had rightly dealt with those conditions which were responsible for the majority of deaths in acute abdominal emergencies, but Dr. Burnell wished to consider some less common, but nevertheless important, conditions. It was common knowledge that one had always to exclude thoracic disease when confronted with what purported to be an abdominal emergency; in this connexion it was necessary to remember that a very high temperature was in favour of thoracic rather than abdominal trouble, with the exception of acute pyelitis, in which the onset might occur with high temperature and rigors.

It was also important to remember that one might have an abdominal emergency with no muscular rigidity or tenderness in the following conditions: (*a*) a patient with thin, flabby muscles such as occur in old people; (*b*) severe toxic conditions, where the neuro-muscular reflex did not function properly; (*c*) in pelvic types of acute appendicitis. There were three acute abdominal conditions in which cyanosis might be marked in the early stages: these were pneumococcal peritonitis, streptococcal peritonitis and acute pancreatitis. So that, provided thoracic disease was excluded, one should always suspect one of these in a cyanosed patient.

Dr. Burnell had not been able to derive much help from the obliteration of liver dulness in the nipple line in diagnosing gas in the peritoneal cavity, but found the obliteration of the dulness in the axillary line of much greater value. In the former case, distended small bowel from any cause tended to be forced up over the liver, and this made it difficult to say whether one was dealing with free gas, or with gas in the intestine.

With regard to rupture of liver or spleen it was important to remember that a considerable interval might elapse between the receipt of the trauma and the actual rupture of the organ. This particularly applied to the spleen, and in one such case which he had treated the interval was seven days.

In epidemics of influenza he had seen patients with high fever, abdominal pain and a tender swelling in the abdomen, but careful examination of the swelling had shown it to be in the rectus sheath, and this condition was not uncommon. He knew of some patients who had been operated on on account of the swelling in the rectus. He could not forbear to mention the necessity of examining the knee jerks in abdominal conditions, as although this seemed very obvious, still patients were being subjected to operation for the gastric crises of tabes.

He finally wished to stress once again the great importance of a carefully taken history in these patients. When one saw a patient writhing in pain, the tendency was at once to feel the abdomen, but without a detailed history this was liable to lead to mistaken diagnosis, and thus to do the patient a great disservice. If such a patient suddenly lost his pain, this was not a matter for congratulation, but rather indicated that some organ, such as a distended gall-bladder or appendix, had ruptured, and the indications for operation became even more urgent than before.

DR. R. G. BURNARD thanked Dr. Lindon for his paper, which was packed with invaluable teaching, especially for the general practitioner. There was one cause of acute abdominal catastrophe that had always to be kept in mind, and that was a ruptured ectopic gestation. Dr. Burnard quoted a case which gave a typical picture of a ruptured gastric ulcer, with extreme shock, board-like abdominal wall, and all the pain located up towards the diaphragm. It was only after the history was carefully elicited that the true cause of the illness was discovered. In regard to obstructed or irreducible hernia, if seen early he had found that if the foot of the bed were raised, and the patient were given double the usual dose of morphine and atropine,

then in about two hours' time the hernia would have slipped back or would be very easily reduced. He had used this procedure many times and had never known it to fail.

DR. A. R. SOUTHWOOD complimented Dr. Lindon on the excellence of his paper; it had been one of the best addresses he had listened to at any meeting of the Branch. Dr. Southwood briefly reviewed, from the standpoint of the physician, several conditions giving rise to acute abdominal pain. He agreed entirely with Dr. Lindon's thesis that it was frequently wise to operate before diagnosis was established. Brilliant accuracy of diagnosis might only be attainable at the price of gloomy certainty in prognosis.

Before operation was decided upon, the most careful consideration should be given to all possible ætiological factors. Dr. Harry Gilbert had mentioned diabetes as a cause of acute abdominal pain. Severe ketosis from any cause might give similar symptoms, and so mislead an unwary surgeon. The gastro-intestinal manifestations of uræmia were sometimes confusing. The severe colicky pains of fireman's cramp and of lead poisoning should also be thought of. The myoclonic form of encephalitis, when it involved the muscles of the abdominal wall, was a rare condition; Dr. Southwood had seen a case, and had advised operation on the mistaken diagnosis of an acute intraabdominal lesion. Intrathoracic lesions often gave abdominal symptoms: pleurisy, pneumonia, and cardiac disease (especially the acute symptoms of coronary occlusion) were all well-known simulators. The pain of the preemptive stage of *herpes zoster* was occasionally severe, but should not mislead a careful observer.

DR. F. R. WICKS said that although it was necessary to stress the fact of excluding the pulmonary lesions in cases of abdominal pain, it was sometimes equally necessary to exclude the presence of an abdominal lesion in cases of definite pulmonary infection, and in such cases not to be content to attribute slight abdominal pain to the lung condition without very careful abdominal examination; this abdominal pain might prove to be a coincident abdominal affection.

He stated that he had reported a case of influenza occurring during an influenza epidemic, where a slight abdominal pain was at first thought to be due to a demonstrable right sided pulmonary congestion, but which on operation twenty-four hours later was found to be due to a gangrenous appendicitis. He also asked Dr. Lindon whether he would consider the appendicitis as a complication of influenza or purely a coincidence.

Research.

THE NATIVES OF THE NORTH-WEST OF SOUTH AUSTRALIA.

PROFESSOR J. B. CLELAND, Chairman of the Board for Anthropological Research of the University of Adelaide, and a member of the expedition, has forwarded to us the following report.

The seventh expedition organized by the Board for Anthropological Research of the University of Adelaide, in conjunction with the South Australian Museum, visited the Musgrave Ranges, towards the north-west corner of South Australia, in August, 1933, for further study of the Australian native. Much of the expense incurred was borne by a fund from the Rockefeller Foundation administered by the Australian National Research Council. The Chief Protector of Aborigines for South Australia, Mr. M. T. McLean, accompanied the expedition.

In the extreme north-west corner of South Australia is an area, 21,900 square miles in extent, which has been dedicated as a reserve for the natives of that portion of the State. In the adjacent corner of Central Australia is a similar reserve, slightly larger in size, extending nearly as far north as Mount Liebig. In the portion of Western Australia adjoining these two reserves is another reserve in that State, also 21,900 square miles in extent. The three reserves form a considerable area, mostly of mulga or sandhill country, and traversed more or less in an easterly and westerly direction by several mountain ranges, notably the Musgrave, Mann, Tomkinson and Petermann Ranges.

The eastern portion of the Musgrave Ranges is outside the reserve, and it was to Ernabella, in this eastern portion, that the expedition journeyed. The main party had been preceded by Mr. N. B. Tindale, Ethnologist to the South Australian Museum, and Dr. Cecil Hackett. During June and July these two, under the guidance of Mr. Alan Brumby, had, on camels, traversed the Musgrave Ranges and entered the Mann Ranges, studying the natives as engaged in their daily avocations, taking measurements, testing the blood groups, and carrying out as much work as the circumstances of travel permitted. Towards the end of this journey the natives met with were directed to go to Ernabella for study by the main expedition. It thus happened that on the arrival of the party at Ernabella, about eighty aborigines, including men, women and children, and belonging to the Jankunzazara and Pitjanzara tribes, were awaiting its arrival, and others came in during its stay, the total number in camp being about one hundred.

The main party left Adelaide on August 3 and journeyed by train to Oodnadatta. The journey to Ernabella, nearly 300 miles north-west of Oodnadatta, was made by motor lorries and a motor car, and occupied three days. The route lay first of all over gibber plains and rises traversed by water courses and with varying amounts of mulga and saltbush. This tableland type of country, in which flat-topped hills are characteristic, their silicified crusts weathering to form the gibbers, was replaced from Moorilyanna onwards by mulga plains of sandy loam, presenting frequently a park-like appearance. From these rocky hills, often granitic, rose at intervals. At the bases of some of these hills deep red sand had accumulated, piling up against the rocks. As rain had fallen in parts, an ephemeral vegetation had in places transformed these picturesque red rises to a dull grey-green, which was soon to be replaced by white as the principal plant, *Myriocephalus Stuartii*, burst into bloom. In addition, between the mulga, open spaces occupied by salt bushes (*Atriplex* and *Kochia*) were not infrequent. Claypans were sometimes seen, and low-lying areas were occupied by tall bushes of giant saltbush. Water courses lined by red gums or box gums were passed at intervals.

The Musgrave Ranges themselves are very picturesque, with long sloping sides and often acute summits. The sides are strewn with rocks and boulders, and between these grow clumps of porcupine grass, the so-called spinifex (*Triodia* sp.). On looking at such a mountain side it will be seen that about equal portions are covered by spinifex and by exposed rocks. In climbing, one is continually pricked by the knitting-needle-like leaves of the porcupine grass as one steps from boulder to boulder. Some of the lower hills may be sparsely covered with mulga or with native pine. Flat valleys run between these ranges, and the abruptness with which the rocky masses emerge from these plains is surprising, as there seems to be little accumulation of *débris* at the foot of the hills. It is along these winding valleys, or glens—and the Scotch term "glen" is singularly appropriate in as much as these mountain masses, especially when enveloped in mist, are strongly reminiscent of the Highlands—that the natives more particularly live. Here game is abundant and water is found in rock holes or in soaks. The ranges provide euros and wallabies, and two large species of *Nicotiana*, used for chewing. Various shrubs and herbs and seeds are also obtainable. To the south of the Musgrave and Mann Ranges there is an extensive area of sandhill country where the water difficulty is acute. The main expedition did not enter this sandhill country. The natives are essentially nomads, passing from spot to spot within the allotted area of the tribe to which they belong, rarely spending any long period in one particular region. This insures that the game and other natural products will not be exterminated, and it would appear that there is a kind of regular succession of places

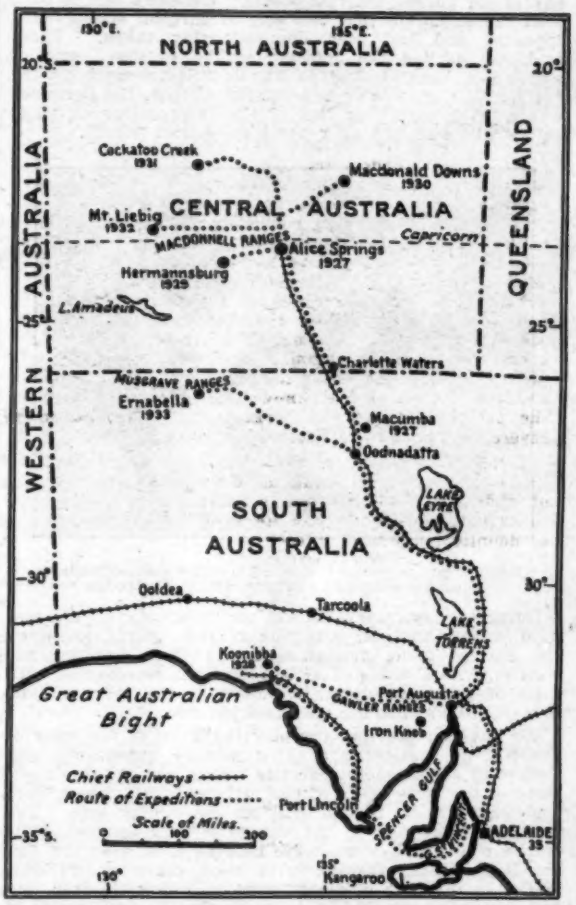
visited by a particular group of people during the course of a year. This raises a difficulty in connexion with the reserves, as in some cases the natural circuit may take the natives outside the reserve and this may eventually lead to trespassing on leaseholds. Of recent years new features have appeared in the food supplies of these regions with the advent of the rabbit and the cat. Both rabbits and feral domestic cats extend throughout this area; the rabbits form a readily accessible source of food, one much easier to obtain than native animals. "Putji kata", as the natives call the cats, being unable to pronounce the "s", are much relished, and during the expedition of Mr. Tindale and Dr. Hackett the natives secured one almost daily. These live in rabbit burrows and prey upon young rabbits, and probably also exact a toll on the bird population and small native mammals. Foxes also have reached this country.

With the nomadic tendencies already referred to, it is difficult to hold together a collection of natives for more than a few days. At first, when the novelty is great and "tucker" is abundant, everyone is very interested and full of energy; at the end of about ten days, however, the natives begin to tire somewhat, become restive, and some of them are sure to want to go for a "walk about". They were fed abundantly morning and evening, during our stay, on heavy damper, boiled wheat (which they liked very much), buck currants (also very much relished), tea and sugar. They soon noticed the absence of meat, and so from time to time sent out their young men or the women, the first to spear wallabies and the latter to dig out rabbits. On Sunday they were given a half-holiday, being fed only in the morning, and were content to forage for themselves for their evening meal. The evening meal is the chief one under natural conditions, and food is not taken on arising, for the simple reason that it is always all eaten the night before.

We found these wandering tendencies beginning to assert themselves towards the end of our stay, and it seems quite impossible to hold for a longer period than about a fortnight any large congregation of natives. There is no doubt that the intensive study made by us during these expeditions enables a very large amount of material to be collected in a short space of time, which otherwise would necessitate months of labour and travelling.

As on previous occasions, each native was given a number and the individual's name, approximate age, genealogical data *et cetera* were entered on a card. With his number painted on the back of his shoulder in duco, the individual passed to the physical anthropologist, who took fifty-three different measurements of various parts, as well as notes on the colour of the skin and the eyes, tribal scars, pathological lesions, the condition of the teeth, and so on. Standard photographs were then taken and the individual was blood-grouped. Plaster casts were obtained of four men and two women, and full busts of four men. Physiological observations were made on certain selected natives, and the general behaviour of the natives and their reactions to various conditions were studied; they were much interested in various toy figures that were shown them. Plants and animals used by the natives were collected and the native names obtained.

The natives soon entered into the spirit of these investigations, though they were somewhat shy during the first day. Thereafter the utmost willingness was always manifested, and any individual accidentally left out temporarily from some part of the routine would either himself, or by one of his fellows, call attention to the omission. Not the slightest difficulty was met with in securing blood by puncturing the ears even of small children, who uttered not a whimper. Still more remarkable is the success obtained in taking face casts and moulds of the chest. The natives grow beards, which in some of the elderly men are quite fine products and evidently much prized, as was shown by the fondling that an old man gave so frequently to his. It was therefore with some hesitation that Mr. Hale endeavoured to secure volunteers for face masks. The person had first of all to be shaved, and what was going to happen had to be explained to him. His face was then covered with coconut oil and vaseline and then wet plaster was applied, and this required much care in the neighbourhood of the eyelids and nostrils. The subject, of course, must keep perfectly quiet for about half an hour for a face cast, and longer for the bust. However, when the matter was explained and it was indicated that a pipe or a red handkerchief would be the reward, volunteers were soon obtained and proved very excellent subjects.



Map of South and Central Australia, showing the various places visited by the anthropological expeditions organised by the University of Adelaide.

The natives live without a stitch of clothing, and this in spite of the temperature falling frequently below freezing point. During our stay it registered 27° F. and 26·5° F. during the night on two occasions. Pools of water had ice on the top, and our clothing was covered with ice crystals. Yet under these conditions the natives, men, women and children, lay absolutely naked in between their small fires. They tend to lie at night in a row, unless it is a family group, with a little fire on each side of the individual, and sometimes one at the feet. The fires are replenished from time to time during the night. Accidents are apt to occur from burning branches, thus explaining the many scars on the body and some examples of injuries to the eyes with resulting loss of sight. The natives always carry fire with them in the shape of burning sticks, and during cold weather may use these lighted brands to keep themselves warm when moving about by waving the stick in front of their bodies. As the result superficial burns are not infrequent.

We found these natives again a very delightful set of people with evidently a high moral code of their own,

which is carefully followed, although doubtless exceptions occur. We found them full of a sense of fun, being very amused at our mistakes in the pronounciation of native words, and yet very polite withal.

On previous occasions we have commented on the fact that we left our gear exposed and tents open, and that there had never been any attempt at interfering with any of our goods. On this occasion the same can be said, with the exception that two sets of surgical scissors disappeared and had been unquestionably taken. These evidently excited the interest of the natives, some of whom had been allowed to handle scissors used by the party. We were unable to ascertain whether the purloiner was a wild native or one of the interpreters or some native who had had an acquaintance with whites.

Knocking out an upper incisor of a young man. Ernabella, Musgrave Ranges. (Photo, Dr. C. Hackett.)

During our stay several wet and misty days were met with, a very unusual condition in these parts, and then the natives took themselves off to their wet weather wurlies, which consisted of leaning branches covered with tufts of porcupine grass, and open on the leeward side, opposite to which a fire was kept burning.

We have on previous occasions referred to the soles of the feet as presenting a thick rubbery appearance suggestive of an old motor car tire. This was again noticed, some of the soles being of this nature, whilst others were somewhat firmer, though still on the spongy side. The arching of the foot in the women is very marked. The tracks made by a woman are said to show that the feet are kept nearly parallel with each other in walking, whilst in the case of males, even in young children, the foot is turned outwards a little, so that the inner side of the great toe is in line with the external malleolus. All the initiated men were circumcised and the urethra was also subincised for half way along its length. Blood enters largely into the ceremonies, and the blood for this purpose is obtained either by venesection from the arm or by stabbing vigorously with a sharp-pointed stick the subincised urethra.

Old scars and burns, and wounds and injuries to the eye from dust and smoke and sparks from fire sticks and penetration by mulga twigs, were seen, but the natives appear almost free from serious disease. The teeth showed much attrition, especially in front, and this is probably due in great part to the use of the teeth for biting sticks in two and sharpening them, pulling out wallaby tendons, and so on. Very little caries was present, but recession of the gums and some pyorrhœa was present in a number of cases.

The following summary indicates more in detail the work carried out by members of the expedition.

Mr. N. B. Tindale registered and numbered each native in turn and secured full sociological and other data on cards correspondingly numbered. It thus became necessary for all other members of the party merely to note the number of any aboriginal dealt with.

Professor J. B. Cleland, assisted by Professor T. Harvey Johnston, ascertained the blood groups of sixty-three natives and cross-tested a number of native sera with native red cells. These individuals were also examined for pathological lesions and ectoparasites. The aboriginal names of a number of plants and animals were obtained and their economic uses, if any. Finger prints and dermatoglyphs were also secured. The findings may be summarized as follows:

Blood grouping with reversing	63 individuals
Cross-testing of native blood	394 tests
Dermatoglyphs of both hands	48 individuals
Finger prints	40 individuals
Examination of stools for parasites	6 individuals
Examination of skin for ectoparasites ..	63 individuals
Pathological, including dental, examinations	55 individuals

Professor C. S. Hicks, assisted by Mr. J. O'Conner, reports that the previous metabolic work at Cockatoo Creek and Mount Liebig showed that the naked aboriginal wasted heat during the cold nights and that as the sun rose the metabolism showed up until in the heat of the day it was up to 20% below the calculated value. Katathermometer values and skin temperatures, as well as respiratory quotient values, indicated that the metabolic adjustment in the native was rapid and elastic, while the low blood pressures recorded, combined with low cardiac rates rising with temperature, pointed to the necessity for making a study of the vasomotor and cardiomotor mechanism under similar conditions of temperature change.

Unfortunately for this work, the weather at Ernabella was poor. The cold night-hot day cycle was replaced by wet weather, high humidity and cold winds. The results obtained represent the work done as occasion suited, and may be summarized as follows:

Twenty-four natives were examined (*a*) in the early morning between six and eight o'clock, as they lay in their camps on the ground with no shelter, and (*b*) during the hottest part of the day, two hot days which occurred during the period being chosen for this purpose.

A young girl adorning herself by inserting the small capsules of a eucalypt into locks of hair. The end of a strand of hair is folded back and jammed into the open end of the "gum nut" with a small stick. She is looking obliquely at what she is doing. Musgrave Ranges. (Photo, Dr. C. Hackett.)

Surface area was estimated (*a*) by the height-weight formula of Benedict and Carpenter, and (*b*) by the sectional measurement method of Du Bois, and Pelidisic measurements were also computed.

The surface temperature was mapped according to Benedict, skin thermometers specially constructed for Sir

Charles Martin being used. Our Cambridge thermostat was broken in transit and the thermocouple could not be utilized. Wet and dry bulb and Kata readings were taken simultaneously with the examination, as were also pulse and respiration rates and blood pressures. Continuous records of pulse volume were made, using the Sahli volumbolograph of latest design. The results indicate, as was hoped, a close correlation between pulse rate, pulse volume and surface temperature and Kata readings. It would appear that the low cardiac rates in the cold observed at Cockatoo Creek were associated with increased output per beat of heart, and that as the temperature rises, blood is transferred to the cutaneous area for cooling and thus removed from the muscle area in accordance with the need for less heat production. The striking feature of the results is the increase in radial pulse volume in an individual of more than four times. This adjustment is rapid and is carried out with ease by young and old. It indicates to some extent the basis of resistance to pulmonary congestive states, and the results may prove of value in functional pathology.

This report is naturally incomplete, as much is yet to be done with the correlations before publication. This hæmodynamic study, however, is a necessary sequel to the metabolic studies previously referred to.

Graded exercise studies were carried out on some twenty-four natives, the effort being twenty knee-bends at the rate of two per second. Recovery of pulse, respiration and blood pressure was studied for four minutes.

The following represents a summary of this part of the work:

48 blood pressure estimations.
48 pulse and respiration checks.
480 skin temperature measurements.
24 graded exercise experiments involving 96 blood pressure and cardio-respiratory tests.
2,400 pulse volume optimal pressure measurements.
48 double checked body temperature measurements.
400 body surface area Pelidisi measurements.
120 Kata thermometer measurements, complete with calculated wind velocity and cooling power.

Dr. H. Gray and Dr. C. J. Hackett measured 61 individuals (males, females and children) for 53 measurements, and on all these individuals made physical observations on the nose, lips, ears, body scars, face shape, hair tracts, dentition, eyes, skin colour, hair *et cetera*.

Group of natives at Ernabella, Musgrave Ranges. (Photo, J. B. Cleland.)

Mr. H. M. Hale undertook the moulding of faces and busts in plaster of Paris, and the "still" photography. He found that, as on previous occasions, the aborigines proved ideal subjects for this work. With some assistance from Mr. Tindale, face moulds of four men and two women, and full busts of four men were secured. The preparation of the busts occupied considerable time, as all hairy areas were of necessity moulded with a plastic compound backed with plaster, and were cast immediately on removal from the body. Data regarding colour of skin, eyes and hair, secured by the anthropometrists, are used when the casts are later coloured.

Seventeen models of animals, made by the aborigines in clay and plasticene, were also moulded for permanent record.

Mother nursing her baby, her hair adorned with the capsules of a eucalypt. Musgrave Ranges. (Photo, Dr. C. Hackett.)

Standard photographs (at least full face and profile) of all natives dealt with were obtained, and numerous pictures were taken of three ceremonies near the camp.

Dr. K. Fry made attempts to interest the natives in Porteus mazes and psychological tests, but found that responses were so casual and distracted that they were of no value. Observations were therefore directed towards obtaining a vocabulary and the study of native customs, beliefs, games and technology. These studies were supported by the cinematography of ceremonies, sign language, hunting, games, the manufacture of spears by adults and children, and the making of wooden vessels and string.

Cinema records of the walking and running gaits of men and women were made, and others illustrate incidents in the daily life of the natives. A new departure was the collecting of plasticene models manufactured spontaneously by the natives. Modelling is not a native art, but the primitive representations which resulted strongly suggest archæological types.

Mr. Norman B. Tindale and Dr. C. J. Hackett report as follows on the field work carried out by them between May 31 and July 30.

We left Adelaide on May 25, 1933. From Oodnadatta we journeyed 280 miles by motor lorry to Ernabella, where there is situated the homestead of Mr. S. Ferguson, a pastoral leaseholder. We were fortunate in securing the services of Mr. A. Brumby, nephew of Mr. Ferguson, who provided an excellent string of camels and the necessary equipment for our journey. A fortnight was spent in studying two groups (134 individuals) of natives who visited Ernabella to trade wild dog scalps. The larger of these groups of natives belonged to the Jankunzazara tribe, of the Musgrave Ranges, but some of the others were eastern Pitjanzara people. One of us (C. J. Hackett) carried out anthropometric observations on 41 natives, and the other, routine sociological records and other data. Some pre-initiation ceremonies in progress were witnessed and recorded. During this period Brumby completed the gathering of equipment at Walta Ijaru'kara, in the Everard

Range, and his native assistants prepared the camp for the August party.

We left Ernabella on June 13 with a string of eighteen camels, carrying food and equipment for two months' sojourn in the reserve. Our itinerary was as follows:

Camp 1.—Mouth of Glen Ferdinand. An early start was made the following morning, but were delayed by camels throwing off their loads. Sixteen natives were encountered during the day's march.

Camp 2.—Upsan Downs. This is newly established as the westernmost outpost of white occupation, and is based upon a well which had been sunk only a few weeks previously. From this place a visit was made to O'walina, an important native dry season water, now included in a pastoral lease. From Mount Woodroffe many native waters were indicated to us, and a preliminary view obtained of some of the country afterwards traversed.

Camp 3.—Made at Erliwanjawanja. Owing to its large capacity, this is one of the important dry season native waterholes on the south side of the Musgraves; it is situated a few miles outside the boundary of the reserve. Sixteen natives belonging to a group who did not desire to proceed further west with us were studied here during a stay of three days. On the fourth day it began to rain shortly after we had commenced loading up. Notwithstanding this, we travelled westwards to Konapandi, a temporary soak and native ceremonial ground, just east of the boundary of the reserve. The arrival from the west of 240 Pitjanzara natives who were gathering for initiation ceremonies, and the continuance of the rain, led us to stay at Konapandi for several days. We were permitted to observe all stages of the initiation ceremony, over a period of several days, on two boys, and were able to photograph all those phases which took place in daylight.

On June 24 we left Konapandi and, travelling west along the south side of the Musgrave Ranges, entered the reserve. Approximately 120 natives followed us to Arukalanda (Camp 5), where the two newly initiated boys had also secretly repaired. After leaving Arukalanda we travelled rapidly for three days towards the Mann Ranges. Forty-one natives continued with us to Camp 6 at Meiti, but the rapid travelling soon caused about half of them to drop behind. A long day's journey brought us to the Caroline (Ulkia, Camp 7), whence we continued in a west-north-westerly direction over sand dunes covered with porcupine grass to a small rock hole called Pudalja (Camp 8) near the west end of the Musgrave Ranges. This rock hole contained barely sufficient water for the natives who were following us. Another day's journey westward along the foothills of the Musgrave Ranges and across the open mallee-covered plains in the direction of the Mann Ranges brought us to a small granitic hill called Pital (Camp 9). This has small native rock holes upon which the natives depend for water when crossing from the Mann to the Musgrave Ranges. Straying camels delayed us here for half a day, and it was dark before we completed our journey to Umbukulu (Camp 10) some miles north-east of Day's Gully (Peltadi). As local water supplies appeared sufficient for several days' stay, we continued our routine anthropometric work, while our native helpers searched for traces of the inhabitants. By long-continued inspection of the horizon and observance of "smokes" they concluded that one group was south-west of Sevenson Peak and that the main Mann Range group had travelled south-west towards the Tomkinson Ranges. Many scattered smokes to the south-east were interpreted as being due to the splitting up of the large groups left at Konapandi and Arukulanda. Having exhausted local water supplies, we moved north-westward along the northern side of the Mann Ranges to Walal (Camp 11), some four miles north of Mount Whinham. The following day we travelled west to Wankarei, north of Mount Charles (Camp 12). We encountered a small family group who were travelling eastwards. They informed us of the whereabouts of others, and messengers were sent to find them. At this camp we were again delayed by straying camels.

From Nankarei we pursued a tortuous course through passes in the massive gneiss ranges to a rock hole (Camp 13), called Analtakutjara, of approximately 120 cubic feet capacity, which is typical of the waters upon which natives rely. After much rock *débris* had been cleared from the approach it became possible to water the camels by pouring the water down the face of the rock into a prepared basin at the foot. Numerous places of importance in Pitjanzara legends were seen during our sojourn in the Mann Ranges. This is an indication that the natives are here wandering in country which has been their own for some generations.

Further wanderings in the ranges brought us, on July 7, to Poka, or Trew Gap (Camp 14), where we settled down to detailed work. A group of some 70 natives soon joined us, chiefly belonging to the vicinity of Malara in the Tomkinson Ranges, and to the western Mann Ranges and the country south of the Petermann Ranges. They proved to be interesting subjects for study. They indicated that a further group of Pitjanzara were moving about west of the Tomkinson Range. Detailed work brought the list of natives studied up to 129. Several *inma laka* ceremonies were witnessed and native legends transcribed. The camels strayed and two were poisoned by eating emu bush (*Duboisia Hopwoodii*).

We left Poka on July 13, accompanied by about 90 natives, who were going to Malara, and crossed to the south side of the Mann Ranges through Trew Gap; cold, boisterous winds blew from the south-west; this made travelling unpleasant. Camp 15 was pitched at Pakiwandi, a native rock hole and soak five miles east of Trew Gap. The following day's journey was made under leaden skies with a cold wind blowing. Leaving the Tomkinson Range people behind us, we continued eastward, passing many important native places, to Kanpi (Camp 16), where there is a rough pass leading between Mounts Edwin and Berry to the north side of the Mann Ranges.

Having parted with all but our own native staff, we left the Mann Ranges and, travelling south-east, crossed broad plains with mallee, porcupine grass and kurrajongs, to Kunamata, west of Mount Kintore, where smokes had been seen. Camp 17 was a dry one in limestone country with porcupine grass, mallee, and kurrajong trees. On the following day we crossed tracks of some fifty natives who had travelled westwards only a few days before. The native rock hole Kunamata is the totem place of the ancestral fig (*ili*) being. A native family met here informed us that the large assemblage at Karapandi had dispersed in family groups in the "sandhill country". We therefore continued our journey eastward along the north side of the Kintore to a small native rock hole (capacity perhaps thirty gallons) called Wiluwiluru. Here we obtained very useful records regarding the methods of utilization of kurrajong seeds as food.

The following day we travelled over wide plains with open kurrajong, mallee and porcupine, and camped in the sandhills west of Mount Crombie. A short trip under rainy skies brought us on the following day to Ulpara (Camp 21), on the northern side of Mount Crombie. Smokes to the nort-east led us across a region of parallel sand dunes towards Penandi, the main rock hole on Mount Harriet, for it was now essential that natives should be gathered for the main party to work upon at Ernabella. A small group of natives whom we met indicated the presence of a large camp of natives south of Mount Crombie. With riding camels and a native boy one of us therefore left Camp 22 in the sandhills south of Penandi and travelled via Itjarano, where a small camp was located, to a small rock hole called Pundi, approximately twelve miles south-south-east of Mount Crombie, where about thirty natives were seen. They had been attracted there by a "dogger" whose camel tracks and camp we observed more than thirty miles within the bounds of the reserve. In the absence of the owner the camp was in charge of a native.

Heavy rain and adverse propaganda prevented our obtaining more than three family groups (seventeen persons). Useful notes on native methods of travel and hunting during rain were obtained during the return to

Camp 23 at Penandi (Mount Harriet). A further excursion on riding camels led to the discovery of another family group of fifteen persons towards Mount Caroline.

On July 25, thirty natives had been gathered, but the same adverse propaganda by the "dogger", who was competing for their services, rendered our endeavours futile, and only seven (five youths and two children) eventually accompanied us across the sandhill region (Camp 24) to Konapandi (Camp 25), where further natives were encountered. Our followers soon increased to twenty-seven, and after native messengers had been sent to all possible native camps, and to Messrs. J. Lennon and P. Connelly, who willingly cooperated with us, our efforts to obtain natives were rendered successful. The rest of the return journey via Erliwanjawanja (Camp 26), Upsan Downs (Camp 27), and the mouth of Glen Ferdinand (Camp 28) was uneventful. We arrived at Ernabella on Sunday, July 30, and after packing specimens and gear for car transport, continued the gathering of rations and preparations for the advent of the larger party, which reached Ernabella on August 7.

The detailed anthropometric work carried out by Dr. C. J. Hackett during the period May 31 to July 30, 1933, may be summarized as follows:

One hundred and twenty-three individuals were dealt with.

Forty-five anthropometric observations were carried out on each individual.

On 117 individuals general physical and anthropological observations were made, for example, eyes, skin colour, hair, nose, mouth, ears *et cetera*.

Eighty-six individuals were tested for blood grouping.

From fifty individuals dermatoglyphs from both hands were taken.

Gross pathological conditions were noted, especial interest being taken in the malady known as "minki" or "larakintja".

The results are now being prepared for publication.

The work of Mr. N. B. Tindale during the period May 31 to July 30, 1933, included:

One hundred and twenty-nine routine sociological data cards were prepared, each involving the obtaining of thirty-three statements. Each individual was photographed.

Two hundred and seven native waterholes and totemic places were localized.

Three native texts, outlines of other legends in English, and the song series relating to ten ceremonies were detailed. Grammatical notes and a vocabulary of 824 words were prepared. The social organization and kinship systems of the Pitjanzara and Jankunsazara were obtained, and one initiation and nine other ceremonies were witnessed. One thousand four hundred and fifty feet of cinematographic film was exposed. Series of animals and plants of ethnological interest and the associated data were obtained. A long series (not yet unpacked) of ethnological objects were secured for the South Australian Museum.

Correspondence.

LOW BACKACHE.

SIR: Years ago, when I was a boy, my father used to give me London *Punch* to read. I always found it humorous; now I read THE MEDICAL JOURNAL OF AUSTRALIA and find the views of many surgeons quite as humorous as some of the things in *Punch*.

I have just finished reading Dr. Vance's paper on low backache, and the remarks contributed by others, and I must say that many of the remarks are extraordinary. For instance, Dr. Ridler says, with the gravity of a Privy Councillor, that "the most common cause of low back pain was tiredness"; that is to say that a symptom, "tiredness", is the cause of a symptom, "back pain"; a most illuminating explanation!

Mr. Goldthwait is reported by Dr. Vance as getting this off his chest: "Pain in the back, or backache, in the first place almost always represents trouble with the structures of the spine and is rarely ever due to disturbance of the viscera"; and yet we wonder at the increase of crime, as we used to say when, as boys, we heard something more ludicrous than usual.

I am afraid that the specialist can seldom regard symptoms without some bias; should he be an orthopædic man, he regards pain as arising directly from the spot where the pain is complained of by the patient; if he should be a gynæcologist, then, as he subsists on concentrated backaches all his life, he knows only too well that backache is more often a symptom of "something afar from the sphere of our sorrow" and at once looks round for the cause. The table contributed by Dr. Edwards shows that of 777 cases of back pain examined by the radiologist, 356 showed no abnormality of spine or pelvis, and so these can be grabbed by the gynæcologist at once, while he leaves the orthopædic man to play with the other 421.

Now were I to draw up a table to indicate the place of origin of the pain causing the backache, I should put them under the following headings: (*a*) muscular, (*b*) kidney, (*c*) colon and sigmoid and rectum, (*d*) appendix, (*e*) ovary and tubes, (*f*) uterus, (*g*) bladder, (*h*) coccyx, (*i*) sacro-iliac joint, (*j*) short leg.

(*a*) We all know the trouble a torn muscle and fibrositis can cause, and how much lumbar pain can come from such, off and on, during life.

(*b*) Kidney. Only those who have walked about with a pair of gouty kidneys dispensing grape shot down the ureter can tell what backache can be. In the days of Matthews Duncan the "aching kidney" was a standing dish, for the aching kidney meant much backache to long, thin women who were always complaining of their lumbar region. Now that the renal artery can be ring-barked of its sympathetic, things are looking up. I operated a week ago on a poor woman who had been the subject of ten operations for aching kidney for ten years. I took the right kidney out of its capsule and ring-barked the renal artery, and she appears easier.

(*c*) Colon, sigmoid and rectum. The ascending colon is a fertile source of backache across the second lumbar vertebra, and the pain is due to the irritation that goes on in the commonest condition that human beings are subject to: chronic colitis, an almost incurable condition. The sigmoid in women often gives rise to backache as it is overloaded. The habit of taking "salts" is the chief cause of the trouble; the patient has a small watery motion and the hard parts are left to stagnate in the sigmoid and descending colon. Murphy told me that on one occasion he had opened the patient for an abscess connected with the sigmoid and found the remains of a Christmas dinner the patient had had three years previously; he identified it because of some seeds that had been eaten only once by the patient, and that was at that particular dinner. The rectum often gives a bad backache when piles are present and the blood clots in one of the injured veins.

(*d*) Appendix. I need hardly mention our old benefactor, the appendix; thanks to his construction out of adenoid tissue, he aches to order, especially after a chill. His particular pain locally is over the lumbar muscles, two inches below the last rib. No wonder the Yanks call him the "abdominal tonsil".

(*e*) Ovary and tubes. I see that Dr. Glissan states that Dr. Worrall says that "most pains in the back of women are not gynæcological". Well, it is never too late to learn, and so let me here tell that 80% of the backaches of women are due to ovary, tubes or downward displacement of the uterus; anyhow, that is what I learnt after operating on some thousands of cases of prolapsus due to rupture of the *levator ani* and other parts. The pain is located just below the pelvic rim behind, three inches from the mid-line.

(*f*) The coccyx is a fertile source of backache when it is affected by that curious condition coccygodynia, first described by Sir James Simpson.

(*g*) Sacro-iliac trouble. Women are certainly subject to very severe backache over the sacro-iliac region, brought

on after strains whilst twisting the body. If treated with adhesive plaster, put on as I have seen such an expert as Dr. Glissan do the job, they get immediate ease and are often well in a few weeks; otherwise they moan and groan for a month or more. Recently I saw an Italian who had been struck behind by a log and was in agony in one sacro-iliac region. I put on an adhesive plaster, but he got no ease, and I proposed to take him to the hospital, where I would have cut down from behind and have fixed a strong screw into his sacrum, and on to that would have fixed strong copper wire, and then I would have pulled that bone into place, for undoubtedly some of these bones get displaced and go back with a "click".

(*h*) This condition is called "lame back" in America. When a woman has one leg a little shorter than the other she often has chronic backache.

Now I was very surprised that the enterprising orthopædic surgeons of Sydney have not trotted out that famous condition known in America as "sliding back". When I visited the Mayo Clinic years ago the North American surgeons were meeting at Rochester and the great discussion was on "sliding back". One famous man after another arose and gave his views, but no two agreed, so William Mayo, who was in the chair, called on me to give my opinion. I knew nothing about the subject, so I kept that to myself, but I observed that it appeared to me that a mistake had been made in the treatment of these cases. All acknowledged they were instances of "sliding back", and so the proper course to be adopted was to hand them over to the Church, because if they suffered from "sliding back" they were obviously "back-sliders"! This ended the discussion.

Yours, etc.,

STEWART MCKAY.

Lismore,
New South Wales,
June 4, 1934.

"EVIPAN SODIUM" ANÆSTHESIA.

SIR: I read with interest in the journal of May 26, 1934, the article by Dr. J. S. MacMahon and Dr. E. G. MacMahon on "Evipan sodium" intravenous anæsthesia, and the purpose of my letter is to extol the value to the general practitioner of this method of anæsthesia.

To the practitioner in a "one-man" district, where an anæsthetist is not always available, and the risk attached to the administration of an inhalation anæsthetic and performance of the operation by the same man is always to be considered, the simplicity and relative safety of "Evipan sodium" should appeal. The only assistant necessary is someone to hold the relaxed jaw forward to maintain a free airway.

The Doctors MacMahon have dealt wtih the technique and pharmacology of the drug and have given a wide range of cases, but I shall just mention my series of ten administered in January and February of this year while resident at the Coast Hospital.

January 25: N.W., aged 23 (M.), circumcision; J.W., aged 60 (M.), incision of ischio-rectal abscess.

January 26: L.K., aged 45 (F.), curettage of uterus and insertion of radium to cervix.

January 29: G.R., aged 56 (M.), incision of inguinal abscess.

February 15: L.L., aged 37 (M.), hæmorrhoidectomy.

February 5: R.H., aged 33 (M.), cystoscopy; S.N., aged 24 (M.), curettage of osteomyelitis of mandible.

February 8: G.R., aged 56 (M.), incisions of abscesses in thigh.

February 11: E.B., aged 47 (F.), incisions of ischio-rectal abscess.

February 12: S.C., aged 69 (M.), amputation of finger.

Very suitable cases would be reduction of fractures, for which I have not so far had the opportunity of using "Evipan", but the muscular relaxation in all cases except the first—a young man of twelve stone, and the amount used only 7·0 cubic centimetres—would have been quite sufficient for the reduction of most fractures.

Another advantage is that the patient is able to return home shortly after regaining consciousness.

R.H. was advised to remain in hospital for one hour after the anæsthetic, but felt so well after half an hour that he refused to stay any longer.

The remainder stayed in hospital for further treatment and all recovered consciousness without excitement, nausea, headache or after-effects of any kind.

G.R. required a second incision later and refused to have any other anæsthetic, as he had had previous unpleasant experiences of ether, so in spite of his poor physical condition, due to chronic toxæmia, 4·0 cubic centimetres were given with satisfaction and no ill effects.

Although it is recorded that over 20·0 cubic centimetres have been given, the average maximum dose is 10·0 cubic centimetres, and less than 10·0 cubic centimetres is satisfactory in most cases.

There are those, and I am of their ilk, who contend that ether is the anæsthetic *par excellence* and that any new upstart in the ranks of anæsthesia is superfluous. But in the above-mentioned circumstances, and considering the mortality of three in many thousands, I consider that "Evipan sodium" should have occasional access to ether's *sanctum sanctorum*.

Yours, etc.,

H. R. HERSCHEL BEATTIE,
M.B., B.S. (Sydney).

Teralba,
New South Wales,
June 7, 1934.

COHESION.

SIR: Your excellent editorial in the issue of June 9, 1934, is very timely. You ask "whether there are potential sources of disharmony or, if not of disharmony, of lack of cohesion". To the first query the answer must be inevitably yes; and if disharmony exists, then lack of cohesion soon follows. What has caused the disharmony? A great amount of it can be attributed to the action of the College of Surgeons. This body has chosen to place itself on a high pedestal and to dictate who can and who can not become its members. In a quiet way it is influencing the university and public hospital appointments. It has not thought fit to endeavour to assist the general practitioner to retain the confidence of his patients, rather the reverse. The medical profession has always regarded the British Medical Association, and through it THE MEDICAL JOURNAL OF AUSTRALIA, its advisers and guardians, but apparently other bodies are attempting to have control. These special divisions may have their uses, and as long as they do not become abuses, well and good. Then harmony and cohesion will remain.

Yours, etc.,

"OBSERVER."

Melbourne,
June 11, 1934.

MEDICAL MEETINGS.

SIR: "Medical meetings are not what they were." "Medical meetings are too pleasantly polite." "Nowadays we see none of the stimulating wordy battles that were a joy to the youthful twenty-five years ago." These are statements in your leading article of June 2, 1934. This evidently means that the fighting spirit of the profession is gone. Our morale is bad. And what may be the cause of this effect?

About twenty-five years ago a then senior member of the profession asked the writer for his experience in regard to his obtaining a hospital resident appointment after graduation and an honorary appointment after that.

The writer had to admit that, with the exception of honours men at graduation, it depended entirely on how many friends one had "at court". But when it came to an honorary staff appointment, even honours at graduation

were not taken into consideration. It was entirely a matter of proving during one's residentship to what extent one could prove to be "pleasantly polite" in the right quarters and to what extent one was prepared not to enter into "stimulating wordy battles" unless very definitely asked to do so.

The old gentleman shook his head and used what was for him some strong language. He said: "What is the profession coming to?" He blamed this "crawling servility", putting together "cause and effect". Is it possible that a generation's servility has destroyed our fighting spirit?

Yours, etc.,

"SENEX."

June 15, 1934.

THE TREATMENT OF CANCER OF THE LIP BY X RAYS.

SIR: In Dr. Molesworth's instructive paper on the treatment of cancer of the lip by X rays (THE MEDICAL JOURNAL OF AUSTRALIA, June 9, 1934) the concluding paragraph is as follows:

> It is also interesting in that my experience supports the contention of the Regaud school, that glands in the neck should not be excised unless palpably enlarged. The old doctrine that glands should be dissected, even if not palpable, in cases of carcinoma of the lip should, I think, be abandoned in the light of modern knowledge and experience.

Both as a resident medical officer and subsequently as assistant to the late Sir Herbert Maitland at Sydney Hospital, I saw a large number of patients suffering from epithelioma of the lip operated on by him, together with block dissection of the cervical glands, apart from my own work.

I noted that often a small epithelioma of the lip showed well marked invasion of the glands, which were not palpable in "thick-necked" patients. On the other hand, palpable glands were often found to be free from growth.

Again, it was not uncommon to have patients admitted in an inoperable condition from metastasis in cervical glands years after local removal of an early epithelioma of the lip, and in later years after possibly unskilful treatment by radium.

If the local condition is successfully treated it is very difficult to get the casual Australian to keep under observation, with the result that invasion of the glands may be missed.

No doubt a big factor in the results obtained by radium, X rays or surgery is the skill of the operator. Dr. Molesworth has had a vast experience in the use of X rays, and is a recognized authority, but, in my opinion, there is a very real danger of X ray treatment of epithelioma of the lip in the hands of those who do not possess the same skill.

The vital question is whether the "safety first" operation taught and practised by MacCormick and Maitland should be completely abandoned in favour of the more modern method of "wait and see", so far as glandular involvement is concerned.

Yours, etc.,

ARCHIE ASPINALL.

137, Macquarie Street,
Sydney,
June 15, 1934.

THE VENOM OF ATRAX ROBUSTUS.

SIR: I was interested to read Dr. Kellaway's article, published in THE MEDICAL JOURNAL OF AUSTRALIA on May 26, on the venom of *Atrax robustus*. He is of the opinion that its toxicity is very slight as far as it affects guinea-pigs after the latter have been bitten by the spider, and this is confirmed by Dr. Mackerras in a letter published in your last issue of the journal.

By chance I happened to be, one afternoon about eight months ago, in the Institute of Medical Research, established in connexion with the Royal North Shore Hospital, and was an eye-witness of an experiment that was being carried out by the Director of the Institute, Dr. W. W. Ingram. A large specimen of *Atrax robustus* was induced to bite the hind leg of a healthy guinea-pig. This occurred at 5.25 p.m. The animal squealed loudly on being bitten. Symptoms of salivation followed soon afterwards, and a few minutes later the animal was in obvious distress from increasing paralysis, death finally taking place, after respiration had been affected, at 5.45 p.m., twenty minutes after the bite had been inflicted.

I was only a casual observer of this experiment, but was greatly impressed at the time at the rapid effects of the toxin, and what has prompted me to state these facts is that they appear to be directly opposed to the findings of both Dr. Kellaway and Dr. Mackerras.

Yours, etc.,

E. D. CLARK, M.B.

Mosman,
New South Wales,
June 18, 1934.

Medical Practice.

IMMUNIZATION AGAINST DIPHTHERIA.

THE Council of the Victorian Branch of the British Medical Association has forwarded the following notice for publication.

As the municipal councils in Victoria are making campaigns against diphtheria by immunization and many inquiries are being made as to what charge should be made by the medical practitioner, the Council of the Victorian Branch has resolved that the remuneration for diphtheria immunization should be at the rate of one guinea per hour for a medical officer of health and two guineas per hour if conducted by a private practitioner. Mileage fees should be charged extra.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on June 11, 1934.

The following degrees were conferred in person by the Chancellor:

Bachelor of Medicine (M.B.) and Bachelor of Surgery (B.S.): Hyman Abramovich, Patrick Joseph McMahon, Naaman George Malouf, Edwin Solomon Alexander Meyers, Roden Crichton Scobie, John Owen Walsh.

Bachelor of Medicine (M.B.): John Rothwell Radcliff.

The following appointments were approved: Mr. G. K. Hughes, B.Sc. (Adelaide) as Assistant Lecturer in Organic Chemistry; Dr. H. F. Wilson as Honorary Demonstrator in Pathology; Miss Gladys Carey, M.Sc., as Acting Curator of the Botany Museum; Mr. J. L. Still, B.Sc., as Honorary Demonstrator in Biochemistry.

THE UNIVERSITY OF TASMANIA.

THE annual commemoration of the University of Tasmania was held at the Town Hall, Hobart, on May 8, 1934.

The degree of Bachelor of Laws was conferred on Mr. Frederick Short, M.B., Ch.M. (Sydney).

Obituary.

OSWALD JOYNT.

WE regret to announce the death of Dr. Oswald Joynt, which occurred at Melbourne, Victoria, on June 13, 1934.

THOMAS BUTLER.

WE regret to announce the death of Dr. Thomas Butler, which occurred on June 20, 1934, at Sydney, New South Wales.

Books Received.

THE 1933 YEAR BOOK OF DERMATOLOGY AND SYPHILOLOGY; 1934. Chicago: The Year Book Publishers. Crown 8vo., pp. 455, with illustrations. Price: $2.25 net.

THE 1933 YEAR BOOK OF NEUROLOGY AND PSYCHIATRY; 1934. Chicago: The Year Book Publishers. Crown 8vo., pp. 471, with illustrations. Price: $2.25 net.

THE 1933 YEAR BOOK OF GENERAL THERAPEUTICS; 1933. Chicago: The Year Book Publishers. Crown 8vo., pp. 464. Price: $2.25 net.

HANDBOOK OF THERAPEUTICS, by D. Campbell, M.A., B.Sc., M.D., F.R.F.P.S.; Second Edition; 1934. Edinburgh: E. and S. Livingstone; Australia: Angus and Robertson. Crown 8vo., pp. 464, with illustrations. Price: 19s. net.

CLIO MEDICA: CHINESE MEDICINE: by W. R. Morse, M.D., LL.D., F.A.C.S.; 1934. New York: Paul B. Hoeber. Foolscap 8vo., pp. 208, with illustrations. Price: $1.50 net.

Diary for the Month.

JULY 3.—Tasmanian Branch, B.M.A.: Council.
JULY 3.—New South Wales Branch, B.M.A.: Council, Quarterly.
JULY 4.—Western Australian Branch, B.M.A.: Council.
JULY 4.—Victorian Branch, B.M.A.: Branch.
JULY 5.—South Australian Branch, B.M.A.: Council.
JULY 6.—Queensland Branch, B.M.A.: Branch.
JULY 10.—Tasmanian Branch, B.M.A.: Branch.
JULY 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JULY 13.—Queensland Branch, B.M.A.: Council.
JULY 17.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 17.—Tasmanian Branch, B.M.A.: Council.
JULY 18.—Western Australian Branch, B.M.A.: Branch.
JULY 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
JULY 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 26.—South Australian Branch, B.M.A.: Branch.
JULY 26.—Victorian Branch, B.M.A.: Council.
JULY 26.—New South Wales Branch, B.M.A.: Branch.
JULY 27.—Queensland Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi and xvii

ALFRED HOSPITAL, MELBOURNE, VICTORIA: Honorary Officers.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Ear, Nose and Throat Surgeon.

CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Honorary Officers, Assistant Resident Medical Officer.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Medical Officers.

ROCKHAMPTON HOSPITALS BOARD, ROCKHAMPTON, QUEENSLAND: Resident Medical Officer.

SCHOOL OF PUBLIC HEALTH AND TROPICAL MEDICINE, SYDNEY, NEW SOUTH WALES: Medical Officer.

THE RACHEL FORSTER HOSPITAL FOR WOMEN AND CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Officers.

TIBOOBURRA DISTRICT HOSPITAL, TIBOOBURRA, NEW SOUTH WALES: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
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